MATERNAL AND FETAL OUTCOME IN EARLY ONSET SEVERE PREECLAMPSIA

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fulfillment of requirements for

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CERTIFICATE

This is to certify that the dissertation titled "**MATERNAL AND FETAL OUTCOME IN EARLY ONSET SEVERE PREECLAMPSIA"** is a bonafide work done by **Dr.K.S.DIVYARADHA** in the Institute of Obstetrics and Gynaecology (Madras Medical College) Egmore, Chennai in partial fulfillment of the university rules and regulations for award of MS degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2011-2014.

Prof. DR.V.KANAGASABAI M.D
Dean
Madras Medical College &
Rajiv Gandhi Govt.General Hospital
Chennai – 3

Prof.DR.MEENA UMACHANDER M.D.,DGO.
Director and superintendent
Institute of Obstetrics and Gynaecology
Madras Medical College,
Chennai – 3.

Prof.DR.D.TAMILSELVI M.D.,DGO.
Guide
Institute of Obstetrics and Gynaecology
Madras Medical College,
Chennai -3

DECLARATION

I solemnly declare that this dissertation titled **"MATERNAL AND FETAL OUTCOME IN EARLY ONSET SEVERE PREECLAMPSIA"** was done by me at Institute of Obstetrics and Gynaecology , Madras Medical College during the year 2011 - 2014 under the guidance and supervision of **Prof.DR.D.TAMILSELVI M.D.,DGO.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.S. Degree in Obstetrics and Gynaecology (Branch -II)

Place :

Date :

Signature of the candidate

Dr.K.S.DIVYARADHA
MS Post Graduate Student
Institute of Obstetrics and Gynaecology
Madras Medical College, Chennai -3

Prof.DR.D.TAMILSELVI M.D.,DGO.
Guide
Institute of Obstetrics and Gynaecology
Madras Medical College, Chennai -3

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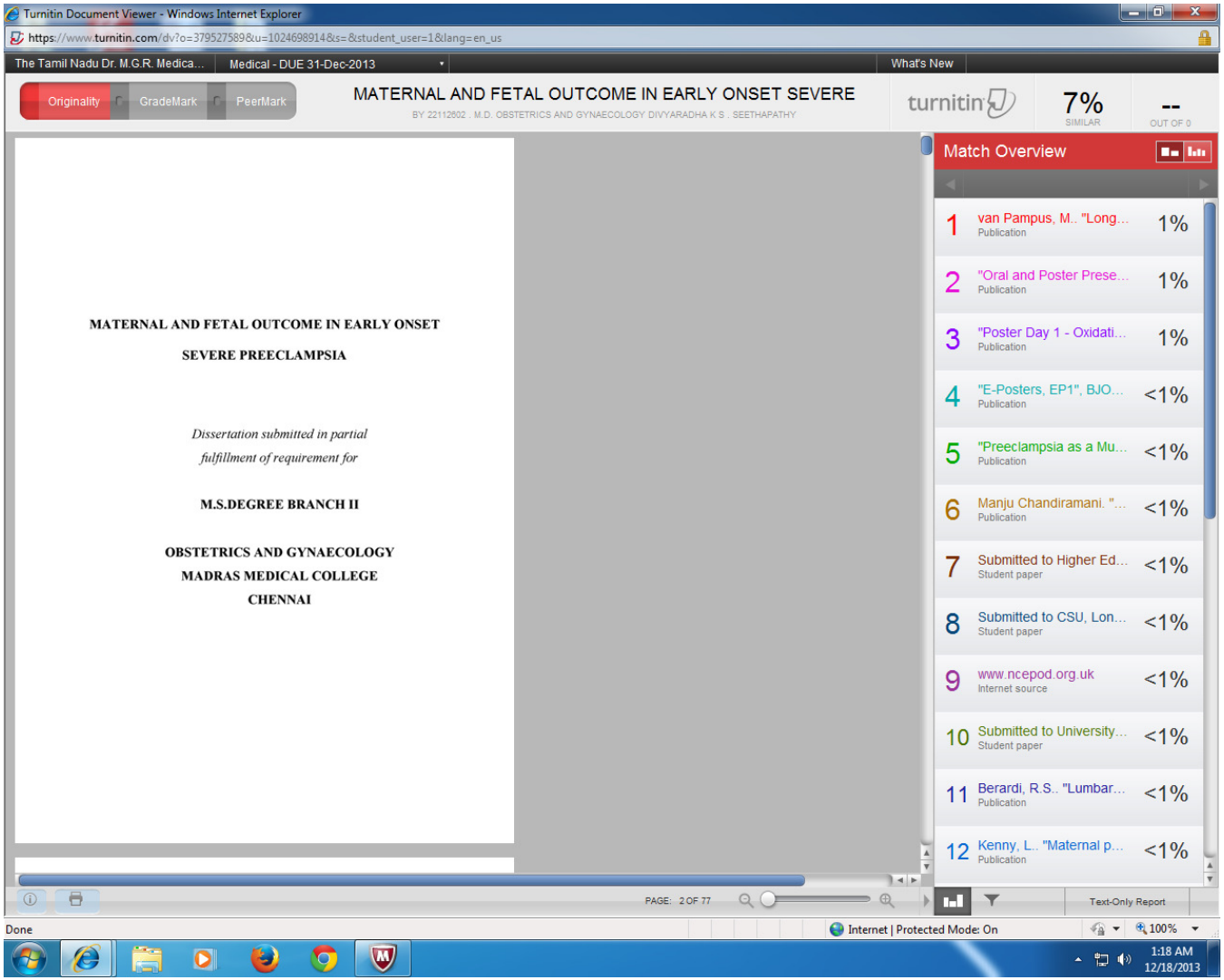
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ABSTRACT

OBJECTIVE:

To study the maternal and fetal outcome in early onset severe preeclampsia and to note the variable factors associated with maternal and fetal morbidity and mortality.

STUDY DESIGN:

It was a prospective analytical study conducted at Institute of Obstetrics and Gynaecology with a sample size of 150 patients who had severe preeclampsia between 24 to 34 weeks.

METHODS:

Patients who met the inclusion criteria were included in the study. Detailed history, examination and investigations were done. Details regarding the treatment and the mode of termination are noted. Follow up of mother and neonate done upto discharge / death.

RESULTS:

More common in Primigravida in the age group of 21-30 years. Mean gestational age at diagnosis was 31 weeks. Most of the women underwent termination after 32 weeks 84(56%).Maternal indication was found to be the most common cause for termination in 128 (85.3%)women, Imminent eclampsia being commonest.104(69%) had caesarean section.32 women in the study group underwent expectant management with maximum prolongation of 25 days.There were no maternal death in patients who were managed expectantly but 4 patients had morbidity and there were 2 fetal mortality and 9 morbidity in the expectant management group. Mean birthweight was 1.43 kg.Out of 150 babies 90(60%) had complications. There were 16 neonatal death and 4 stillbirth with perinatal mortality rate of 30%. 58 (38.6%) women of the total study group experienced complication.Eclampsia and abruption were higher.There were 6(4%) maternal death. Mean postpartum hospital stay was 16 days.

CONCLUSION:

Early onset severe preeclampsia is associated with significant maternal and fetal complications Decision regarding termination of pregnancy has to be taken based on both maternal and fetal factors. Termination should be done irrespective of fetal maturity in case of severe uncontrolled blood pressure with

complications. In selected cases expectant management in a tertiary care centre limit the impact of serious maternal and fetal complications.

KEY WORDS:

Early onset severe preeclampsia, Imminent Eclampsia, Expectant management,Maternal outcome, Fetal outcome, Perinatal mortality.

TABLE OF CONTENTS

INTRODUCTION

Preeclampsia is a multisystem disorder involving placenta, kidney, liver, blood, cardiovascular and neurovascular system, occurring exclusively during pregnancy whose etiology is not known. It occurs in approximately 5-7% of pregnancies. It is an important cause of morbidity as well as mortality in both the mother and fetus.

Preeclampsia is described as a rise in blood pressure and proteinuria which is of new onset, occuring after 20 weeks of gestation. It is described as severe preeclampsia if there is substantial increase of blood pressure and proteinuria or the occurrence of symptoms due to end organ damage. Preeclampsia is considered early onset if elevation of blood pressure and proteinuria occur before 34 weeks of pregnancy. Hypertension and its complications is ranked third as a leading cause of maternal mortality, responsible for over 17% of maternal deaths [1,2,3] .Even in developed countries, women still die from pre – eclampsia and eclampsia.

There is increased risk of acute renal failure, cardiovascular and cerebrovascular complications, abruptio placenta, disseminated intravascular coagulation and even maternal death[1]. So, early

diagnosis and close monitoring in preeclampsia plays a vital role in preventing its complications.

In case of early onset severe preeclampsia, there is progressive deterioration in the health condition of mother and also high morality in the fetus[2,3] during the perinatal period. Delivery the fetus is considered the only way to revert all these complication. So, termination of pregnancy is needed if there is fetal distress, in case of multi organ dysfunction or if the gestation age reaches 34 weeks. But, prematurity due to early termination causes high perinatal morbidity and mortality[1,5,6]. Accelerated fetal lung maturation does not occur in preeclampsia[8] however expectant management to prolong pregnancy can be deleterious to the mother[7,9]. Hence, potential benefits attained for the fetus should be weighed against the potential dangers that occur to the mother.

AIM OF THE STUDY

1) To study the maternal and fetal outcome in early onset severe preeclampsia.

2) To note the variable factors associated with maternal and fetal morbidity and mortality.

REVIEW OF LITERATURE

HISTORICAL ASPECTS:

Pre- eclampsia is a complex disease often labeled as “disease of theories” due to numerous schools of thought.

Eclamptic convulsions have been recognized in ancient Chinese, Indian, and Greek literature dating back 4000 years.

Hippocrates around 400 BC stated headache accompanied by convulsions during Pregnancy was considered bad.

Bossier de saurages (1739) introduced the word Eclampsia and differentiated eclampsia from epilepsy.

Demanet (1797) recognised swelling in eclamptic women. Pierre Rayer (1840) discovered protein in urine while John Lever (1843) reported Proteinuria was specific Pre-eclampsia.

Vasquez and Nobcourt (1897) discovered eclamptic hypertension.

Chesley (1984) said Sensory stimuli were reduced by keeping the patients is dark quite room. Horn (1906) first used magnesium sulphate to manage pre-eclampsia – Eclampsia.

Lazard and Dorsett (1920) popularized the parental use of magnesium sulphate only in 1990. Major studies demonstrated the superiority of magnesium superheat over other anticonvulsants.

In 1967, Robertson and Brosens described structural changes of the utero placental unit in pre-eclampsia.

MAGNITUDE OF THE PROBLEM:

Pre- eclampsia has a prevalence rate of 2.3%[12]. 50% of the women with severe pre-eclampsia present at less than 34 weeks of gestation. Delivery is mandatory either for maternal or fetal reasons within 24 hours of admission.

CLASSIFICATION:

National High Pressure Education program 2000 classification is as follows:

- Gestational Hypertension
- Pre eclampsia
- Eclampsia
- Superimposed Pre eclampsia
- Chronic Hypertension

DEFINITIONS

PRE-ECLAMPSIA

Pre-eclampsia is defined as rise in systolic pressure of 140 mmHg or greater or diastolic pressure of 90 mmHg or greater that develops for the first time in pregnancy after 20 weeks of gestation in atleast two occasions accompanied by proteinuria.

Proteinuria defined as 0.3g or more of urinary protein per 24 hours or persistent 30 mg/dL (1+ dipstick) in a random urine sample.

Severe Pre-eclampsia:

Severe pre-eclampsia is defined as hypertension (diastolic blood pressure ≥ 110 mm Hg) with proteinuria (≥5gm in 24 hours urine specimen or ≥ 3+ on a dipstick).

Early onset severe pre- eclampsia:

When severe pre- eclampsia develops prior to 34 weeks of gestation[14].

ACOG CRITERIA FOR SEVERE PREECLAMPSIA:

Severe Pre eclampsia is considered in the presence of one or more of the following

- Blood pressure- systolic of 160 mmHg or greater or diastolic of 110 mmHg or greater on atleast two occasions, 6 hours apart.

- Proteinuria of 5gm or more in 24 hours urine sample or 3+ or higher on atleast two random urine samples collected 4 hours apart
- Oliguria (<500 ml in 24 hours).
- Pulmonary edema or cyanosis.
- Cerebral or visual disturbances.
- Thrombocytopenia
- Liver dysfunction
- Epigastric Pain
- Fetal growth restriction.

RISK FACTORS:

The most predictive risk factors of pre- eclampsia were mean arterial pressure and Parity. The risk with mean arterial pressure was 8% when < 75mm Hg and 27% if > 85 mm Hg. Risk was 26% is nulliparous where as 17% in parous patients[15].

There is increased risk of pre- eclampsia in women with periodontal disease are and systemic inflammation early in pregnancy with (CRP $\geq 75^{th}$ percentile)[17].

Recent study demonstrated a significant association between maternal thrombophilia and severe preeclampsia[18].

Maternal and paternal genetic factors increase the risk of pre eclampsia.

Pregnancy associated:

- Chromosomal abnormalities
- Hydatidiform mole
- Hydrops fetalis
- Multiple gestations.

Maternal specific:

- Primi parity
- Age < 20 & > 35 Yrs.
- Previous pre-eclamptic
- BMI > 35
- Family history of pre-eclampsia.
- Gestational diabetes or pre-existing diabetes.
- Chronic hypertension.
- Nephropathy.
- Thrombophilias.

Paternal Specific:

- First time father
- Previously fathered a pre-eclampsia pregnancy is another woman.

ETIOPATHOGENESIS:

Various theories have been proposed in the causation of pre eclampsia.

- Abnormalities in the trophoblastic invasion.
- Genetic predisposition
- Role of vasoactive agents
- Immunological theory
- Oxidative stress and inflammatory changes
- Endothelial factor.

Abnormal trophoblastic invasion:

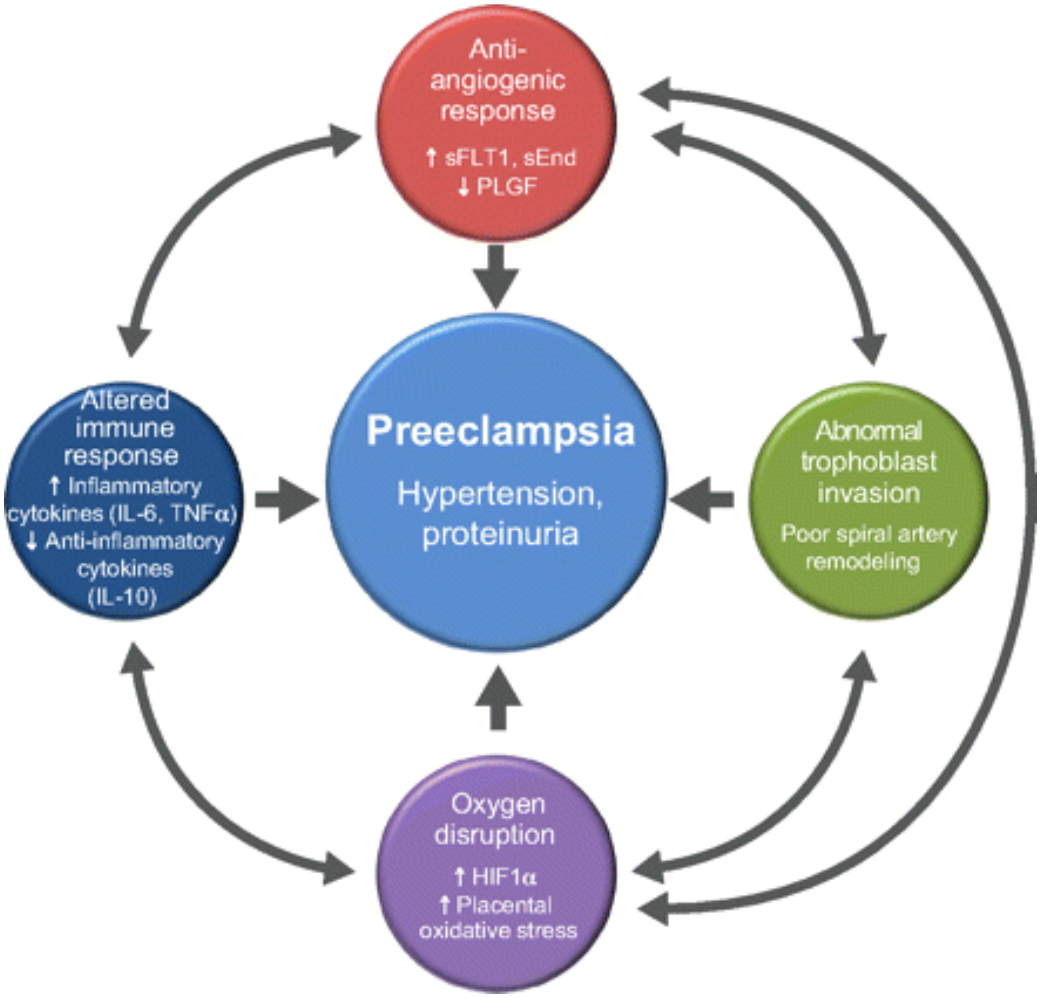
Normally the uterine spiral arteries are invaded by endovascular trophoblasts. In pre-eclampsia there is incomplete trophoblastic invasion[22]. In a recent study of placental morphology, placentas from early onset (<34 weeks) and late onset (>34 weeks) were studied. An abnormal placental morphology was found in early onset, whereas placentas from late onset disease were morphologically similar to those from same gestational age controls.

Dewolf et al examined the arteries from utero-placental implantation site. They observed early pre- eclamptic changes which include endothelial damage, proliferation of myointimal cells, insudation of plasma constituents into vessel walls and medial necrosis. These changes results in diminished placental perfusion, leading to pre-eclampsia syndrome.

Genetic Predisposition:

A multi factorial inheritance has been reported.

An association between HLA DR4 and protienuric hypertension has been reported.



Immunological theories:

These theories are based on incompatibility at the feto-maternal interface. There is disruption of equilibrium between fetal genotype and maternal immune responses required for normal placentation. [23]

- Absence of blocking antibodies - There is an imbalance between the ratio of fetal antigenic load and maternal blocking antibodies. This might be due to excess fetal antigen load

(hydatiform mole, multiple gestations) or absence of blocking antibodies (as in first pregnancy).

Role of vasoactive agents:

In pre-eclampsia there is an increase in Thromboxane A2 and decrease in prostacyclin (PGI2) and prostaglandin E2 resulting is vasoconstriction[24].

Endothelin – 1 which is a potent vasoconstrictor is found to be increased in pre- eclampsia[25].

Vascular endothelial growth factor causes vasculogenesis and important for microvascular permeability. It is found be increased in pre- eclampsia[26].

Endothelial factor:

Endothelial cell dysfunction is a major factor in the pathogenesis of pre- eclampsia.

Nitric oxide which is a potent vasodilator is decreased in pre eclampsia[27].

Elevated homocysteine level in early pregnancy increases risk of pre eclampsia.

Inflammatory change and oxidative stress:

Pre- eclampsia occurs due to extreme stage of activated leucocytes in the maternal circulation. Deciduas when activated

releases leukotreines, TNF Alfa which result in oxidative stress in the body.

There is release of oxygen free radicals due to oxidative stress in pre- eclampsia which result in

- Release of lipid laden macrophages
- Damage to endothelium
- Activation of micro vascular coagulation
- Increased capillary permeability.

PATHO PHYSIOLOGICAL CHANGES:

Hemodynamic Changes:

In Pre-eclampsia,

a) Cardiac preload decreases due to generalized vasospasm[22]
b) Cardiac after load increases due to increase in vascular resistance.
c) MAP is reduced as there is increase is peripheral vascular resistance.

Hematological Changes[22]:

a) Thrombocytopenia – occurs in 10 to 25 % of women having pre-eclampsia. It is probably immunologically mediated or because of increase in platelet deposition at the endothelial damage site.

b) Hyper coagulation is pre-eclampsia is due to

- Increased activity of intrinsic factors.
- Increased platelet aggregation.
- Increased thrombin – Anti thrombin ratio.
- Decreased anti thrombin III and fibrinogens.

Endocrine changes:

In Pre-eclampsia there is suppression of renin- angiotensin - aldosterone axis and sodium retention is increased[22].

Fluid Changes:

Extra cellular fluid volume increases as a result of endothelial damage leading to proteinuria and edema[22].

PREDICTORS OF PRE ECLAMPSIA:

1. Mean arterial pressure is better than systolic or diastolic blood pressure as a predictor for pre eclampsia. The mean arterial pressure during first trimester is elevated in women who develop pre-eclampsia[99]
2. As a marker of early onset severe pre-eclampsia, reduced level of placental growth factor (PIGF) in first trimester is most sensitive as well as specific.

3. Uterine artery Doppler evaluation is combination with soluble fms-like tyrosine kinase (SFlt – 1); placental growth factor (PIGF) and Angogenic factors may be useful as a second trimester screening test for early onset pre- eclampsia.
4. Soluble Endoglin (SEng) is elevated in pre- eclampsia. Combination of Sflt – 1 and SEng is a good predictor of early onset pre- eclampsia in second trimester with 100% sensitivity.
5. Serum activin A and inhibin A are elevated before 20 weeks in women developing early onset revere preeclampsia.
6. Higher HCG, MSAFP, lower MSUE3 are significant markers of early onset severe pre- eclampsia[93].
7. Placental oxidative stress is associated with early onset pre-eclampsia. There is an increase in PAI – 1 / PAI - 2 ratios which suggest a stronger link between early onset pre- eclampsia and a dysfunctional placenta.
8. Women with several pre- eclampsia have higher plasma concentration of adiponectin.
9. Total 25 – hydroxy vitamin D (25 – 0H – D) level is decreased is women developing early onset severe pre-eclampsia. To understand the impact of vitamin D on pregnancy outcomes, further studies are needed.

Maternal Outcome:

Women with Severe Pre-eclampsia are at increased risk for abruptio placenta, acute renal failure, disseminated intravascular coagulation, cerebral haemorrhage, pulmonary edema, circulatory collapse[28].

Murphy and stirrat[29] studied 71 preeclamptic women with gestational age less than 30 weeks and reported 21% had developed HELLP syndrome, 15% had abruptio placenta, 13% had renal failure and 1.4% eclampsia but no maternal mortality was observed.

Al –Mulhim et al reported that the commonest complication to be abruptio placenta.

Perinatal Outcome:

Perinatal outcome is usually dependent on one or more of the following:

1. Gestational age at the time of delivery.
2. Gestational age at the onset of pre eclampsia.
3. Present of multiple gestation.
4. Severity of the disease.
5. Presence of other medical disorders.

Pre-eclampsia accounts for more than 40% of pre-mature deliveries[31] and there is a substantial increase in the risk of low birth weight and SGA babies [32,33,34,35,].

In a study performed by **odegard et al[36]** compared 307 live singleton born to pre eclamptic women to 619 controls, pre-eclampsia and severe preeclampsia were associated with a 5% and 12% reduction in birth weight respectively, and birth weight was 23% lower than expected.

Magee et al[37] in a multi centric retrospective study found out that 16% of pre-eclampsia pregnancies being complicated by birth weight less than third percentile.

Very low birth weight (VLBW less than 1500 grms) and extremely low birth weight (ELBW, less than 1000 grms) babies often require re-admission to hospital in the first two years for respiratory infections[38].

EXPECTANT MANAGEMENT:

There have been 15 non randomized non controlled trials to assess the benefits of expectant management in pregnancy between 24 to 34 weeks and showed an average of 10 – 14 days prolongation of pregnancy in cases of severe preeclampsia without increase in

maternal morbidity but these trials are not reliable as they lack randomization[41].

However, since 1990 only one maternal death has been reported in literature among 1677 women who underwent expectant management[40].

RCOG AND ACOG RECOMMENDATIONS:

AIM:-

To prolong pregnancy in severe preeclampsia women remote from term in order to improve perinatal outcome without increasing maternal morbidity or mortality.

PLACE:-

Management is best accomplished in a tertiary care setting with senior obstetric consultants, obstetrician gynecologists trained in management of high risk pregnancy, experienced staff. Candidates depend on a number of factors like maternal and fetal conditions as well as gestational age[42]. There is no place for expectant management in gestational age <26 weeks as there is increase in perinatal and maternal morbidity and mortality[43].

Expectant Management in IUGR: - studies have showed that in women with server pre-eclampsia at 24 – 33 weeks IUGR is associated with high risk of fetal mortality but does not cause maternal

complications[47] and may benefit from prolongation of pregnancy beyond 48 hours that is required for the action of steroids.

Expectant management in HELLP Syndrome:

Current evidence demonstrates that women with HELLP syndrome should not be managed expectantly. Vaginal or caesarean delivery should be pursued as appropriate. Antenatal steroids may be given concurrently.

TERMINATION OF PREGNANCY:

Termination is needed is the following conditions.

FETAL:-

Severe IUGR

Non reassuring fetal surveillance

Oligohydromnios

Maternal:-

Abruptio placenta

Platelets < 1 lakh

Progressive detoriation of liver and renal function

Signs and Symptoms of imminent eclampsia.

GUIDELINES FOR MANAGEMENT

FETO – MATERNAL MONITORING:

During initial assessment, blood pressure should be measured every 15 minutes till the woman is stabilized and later to be checked every 30 minutes.

Later measurement of blood pressure should be done at least four times per day.

CBC with platelet count, LDH,AST,ALT repeated everyday and more frequently in case of abnormalities.

If platelet count is above 1,00,000 cells/dL, clotting studies are not needed.

Input and output charting is vital in monitoring fluid balance, especially in the immediate postpartum period.

ASSESSMENT OF FETUS:

Fetal Monitoring includes fetal heart rate monitoring with cardiotocography which gives information regarding well being of the fetus. Continuous fetal monitoring should be done in women in labour. Fetal biometry, umbilical and cerebral Doppler and liquor volume should be measured for further assessment. Delivery time can be assessed based on serial measurements. Investigation with Doppler

evaluation using absent or reversed umbilical artery (UA) diastolic flow improves neonatal outcome [50] . Its serial measurements can optimize time of delivery.

NON PHARMACOLOGICAL APPROACHES

Although no study or clinical trial which clearly gives the data about the use of restricted activity in severe preeclampsia, but it is considered good clinical practice to restrict activity and hospitalize these patients.

ROLE OF ANTI – HYPERTENSIVES:

Anti hypertensive treatment should be started when there is elevation of blood pressure to severe range (>160/110mmHg). With severe disease, anti hypertensive treatment should be considered even in lower measurements. For acute management, oral or intravenous labetolol, oral nifedipine or intravenous hydralazine can be used.

Angiotensin converting enzyme inhibitors, atenolol, diuretics and angiotensin receptor blocking drugs are avoided.

Nifedipine is used orally not sublingually. Randomized trails doesnot support the general consensus that treatment is required when blood pressure is higher than 170/110mm Hg[52]. However it is desirous to prevent vascular damage as a result of uncontrolled

hypertension. A lower threshold of 160 mm Hg systole is suggested by enquiries into maternal death.

ROLE OF MAGNESIUM SULPHATE:

Magnesium sulphate remains the anticonvulsant of choice in any kind of set up. Magnesium sulphate has been shown to decrease the progression of pre – eclampsia to eclampsia by more than fifty percent [44].

Pritchard regime is usually followed, which include 20ml of 20% magnesium sulphate (which is equivalent to 4gm) is given as a loading dose by intravenous route over 10 minutes. Later 10ml of 50% magnesium sulphate (which is equivalent to 5gm) by intramuscular route in alternative buttock given every four hours as maintenance therapy [45].

If convulsions recur, further addition of 2 to 4gm is given by intravenous route over 5 minutes. Therapeutic level of 4-6 mg is to be maintained.

Before administrating the subsequent doses, the women should be checked for

- Urine output at least 100ml over the last four hours.
- Knee jerk
- Respiratory rate of atleast 16 per minute.

If any of the above parameters are found to be abnormal, the subsequent dose should be delayed till attainment of normal status. In case of toxicity, 10ml of 10% calcium gluconate (1gm) is given by intravenous route over 10 minutes and oxygenation provided.

In case of persistence of convulsions patient should be transferred to intensive care unit and if necessary maintained with intermittent positive pressure ventilation.

ROLE OF STEROIDS:

It is a well known fact that antenatal glucocorticoids decreases neonatal morbidity as well as mortality in a woman with severe disease at or less than 34 weeks of gestation age [46].

Previously, it was uncertain whether steroids were efficacious in severe pre-eclampsia less than 34 weeks of gestation. However, a prospective randomized trial showed that there was significant reduction in the incidence of respiratory distress syndrome (RR, 0.53; 95%CI, 0.35 to 0.82) in the Steroids group (receiving betamethasone) against the control group (receiving placebo)[46] Thus steroids help in fetal lung maturity and should be used for at least 48 hours. Their use has also been reported in cases of HELLP syndrome as they prolong pregnancy [58] and lead to resolution of hematological and biochemical

abnormality. But their role in reducing morbidity is uncertain according to another study [57].

FLUID BALANCE:

Large majority of women with mild pre – eclampsia have expanded intravascular volume. Hence to avoid fluid overload, fluids should be restricted to about 1ml / kg/ hour or 80ml / hour.

Inappropriate fluid management has often been associated with pulmonary edema which has attainted significance as an important cause of maternal death [53].

Fluid expansion provides no benefit [59]. While fluid restriction have showed good outcome[60]. Fluid restriction is inappropriate in case of maternal hemorrhage or oliguria.

MODE OF DELIVERY:

There are no randomized trails conducted for comparing the ideal method for delivery in pre-eclamptic women.

Vaginal delivery should be attempted in all women with mild pre-eclampsia and in most women with severe pre-eclampsia, provided there is no other indication for caesarean section [5]. Pre-eclampsia as such is not an indication for caesarean section.

In a study of **Gofton et al** in 2001 showed that induction of labour and caesarean section in women with gestational hypertension is twice as

those in the control group and were also similar to pre-eclampsia and chronic hypertension. But, the study did not show any differentiation between mild/severe and early/late of gestation.

Induction of labour with prostaglandins will increase the chance of vaginal delivery. Throughout labour, assessment and anti hypertensives should be continued.

POSTPARTUM MANAGEMENT:

Postnatal close monitoring should be done for women with severe disease . The period of hospital stay is unclear as eclampsia can occur even after 4 weeks postnatal. However after fourth postpartum day, incidence of eclampsia decrease [54].

Inpatient management for four or more days will be required for most women with severe disease after delivery.

Anti – hypertensive treatment should be continued and tapered stepwise after delivery. It can take even three months for blood pressure to return back to normal. No particular anti – hypertensive is recommended. But α- methyl dopa is avoided due to its side effect, like depression. Nifedipine, labetolol, enalapril and atenolol are being used currently in breastfeeding women.

Women should be further investigated in case of persistence of high blood pressure and proteinuria.

PROPHYLAXIS:

In 2003, **Duley et al** found that there is decrease in risk of pre – eclampsia by 19% and 16% reduction in fetal and neonatal deaths with the use of aspirin . 75mg aspirin daily started after first trimester may be offered as a prophylaxis for women at high risk of developing hypertension, (e.g., abnormal uterine artery Doppler, previous pregnancy with pre eclampsia, pre – existing hypertension, etc.,) [63, 64].

A small randomized trial (**Chappell et al**) demonstrated a decrease in pre – eclampsia incidence in women using vitamins C & E. However pre – eclampsia prevention using antioxidants require larger trial for assessing the safety and benefits of this treatment.

Systemic review of 11 randomized controlled trials (**Atallah et al, 2002**) shows that with calcium, there is significant decrease in risk of hypertension and also pre – eclampsia.

***LONG TERM OUTCOME AFTER PREECLAMPSIA*.**

CARDIOVASCULAR DISEASE AND HYPERTENSION:

Higher are the chances of developing cardiovascular diseases and / or chronic hypertension later in life in a woman with history of pre – eclampsia.[67, 68].

In 1995, nissel described that women appear to have chronic hypertension 7 years after a pregnancy complicated by hypertension when compared with a control group [67].

Wilson et al [69] reported that there is association between pregnancy induced hypertension and hypertension related diseases in later life, like stroke.

Irgens et al[70], in the study conducted in Norway with 6,26,272 live births between 1967 and 1992, showed that there is 8.12 times higher risk of death due to cardiovascular disease in women with preterm delivery and pre eclampsia than in the reference group.

Sattar and Greer [71] in 2002 suggested that there is high risk for cardiovascular disease in later life in a woman with adverse pregnancy outcomes and described that both may have common mechanisms [71].

RENAL DISEASE:

There is frequent association of pre – eclampsia in a women with pre – existing renal diseases [72]. Post partum renal biopsy studies showed the classic renal lesion of pre – eclampsia e.g., glomerular endotheliosis. Also lesions like nephrosclerosis or other abnormalities were found in primigravida at a rate of 9.2% and in multigravida at a rate of 37% [68].

Reiter et al, found that only 2.3% women with pre – eclampsia had renal disease in their follow up between three months and six years [72]. Jacquemyn et al found that there is no difference in renal dysfunction between women with HELLP syndrome followed up for 5 or more years and control group who were normotensive during their pregnancy [73].

Another report which studied the long term renal function in women who had pregnancy with pre eclampsia or HELLP syndrome described that there is no effect of pre – eclampsia on long term renal function [74].

HEPATIC DISEASE:

Liver is not primarily involved in pre – eclampsia while liver involvement is observed in only 10% of women with severe pre – eclampsia . One of the severe manifestation of pre – eclampsia is HELLP syndrome.

The hepatic findings in HELLP syndrome consist of focal parenchymal necrosis and periportal necrosis [75]. The most likely reason is hepatic ischemia which results in deposition of fibrin in the hepatic sinusoids. In the hepatocellular necrotic areas, there is infiltration of red blood cells and inflammatory cells. Though there is

considerable damage to the liver parenchyma, it usually recovers within six weeks of postpartum period, which results in regeneration of parenchyma of the liver.

As a result of subcapsular hematoma, there may be rupture of the liver capsule which occurs very rarely but is a severe life threatening complications due to HELLP syndrome. There is complete recovery of liver function in those who survive this complication and in some women, there are reports of successful pregnancy also. The recurrence risk is however unknown[76].

BRAIN LESIONS:

The pathophysiology of neurological disturbances in case of pre–eclampsia is least understood. The most frequent location of specific lesions are the parieto – occipital lobes which is also the anatomic location of petechial hemorrhages and infarction. While most of these lesions were found to be reversible, others were reported to be permanent. Utilizing the modern imaging methods, the lesions were found to be ischemic with resulting small infarcts in the brain i.e., gliosis. Long term follow up of neurological functions in these patients is currently under investigation.

SIMILAR RISKFACTORS OF PRE – ECLAMPSIA AND ATHEROSCLEROSIS:

In a woman with pre – eclampsia and / or HELLP syndrome, there is enhanced risk for venous and arterial diseases [77].

Even after adjustment for other risk factors of coronary artery disease. Pre – eclampsia is found to be an independent risk factor [72]. With a strong family history of cardiovascular disease, more likely is the chance to develop hypertension of pregnancy and pre–eclampsia[78]. There is high chance of preterm birth and intrauterine growth retardation when associated with maternal stress during pregnancy.

The only exception is smoking which is protective against pre – eclampsia where as it is a risk factor for cardiovascular disease. Pre – eclampsia, preterm delivery, low birth weight and a combination of these factors can be a first manifestation in case of atherosclerosis.

Bogges et al [80] reported that there may be transient translocation of oral microbial flora to the utero- placental unit of pregnant women with periodontal disease, leading to oxidative stress in early pregnancy or inflammation of placenta, which result in clinical manifestations of pre – eclampsia and produced placental damage.

RECURRENCE RISK:

The recurrence risk of developing hypertension in subsequent conception is about 20-50%, while that of developing HELLP syndrome is about 2 to 6%.

The major predictors of subsequent conception are gestational age and birth weight. The gestational age will be longer by 5 weeks and birth weight 1300 to 1400g more in the subsequent conception independent of the recurrence [81].

PSYCHOSOCIAL CONSEQUENCES IN PRE – ECLAMPSIA AND HELLP SYNDROME:

There is an increased risk of post traumatic stress disorder (PTSD) as a result of the unexpected complications that occurs during pregnancy. PTSD manifestations include sleeplessness, irritability, exaggerated startle response, avoiding situations, increased arousal, re-experiencing the event,etc.,

Engelhard [82] reported that pre – eclampsia is a primary cause but not an exclusive cause for PTSD which results from concomitant preterm delivery.

MATERIALS AND METHODS

STUDY DESIGN:

Prospective Analytical Study

SETTING:

Institute Of Obstetrics And Gynecology
Egmore
Chennai-8.

SAMPLE :

150 patients with pregnancies complicated by EARLY ONSET SEVERE PREECLAMPSIA at IOG for a period of one and a half years (2012-2013).

INCLUSION CRITERIA:

1. Gestational age >=24weeks and < 34 weeks
2. Diastolic BP >= 110 mmHg
3. Proteinuria >= 3+

with any of the following

4. Persistent headache
5. Blurred vision
6. Eclampsia
7. Elevated liver enzymes
8. Low Platelets
9. Abruptio Placenta
10. Oligohydomnios
11. IUGR

EXCLUSION CRITERIA:

1. Gestational age < 24weeks and > 34 weeks
2. Preexisting chronic renal and hepatic disease.
3. Idiopathic hemolytic anaemia
4. Idiopathic thrombocytopenic purpura
5. Epilepsy

METHOD:

All patients who meet the above mentioned criteria were included in the study and patients were admitted in the intensive care unit .

Patient's detailed history, symptoms and signs of severe preeclampsia ,imminent eclampsia were noted.

General and Obstetric examination was carried out.

Urine investigation and PIH profile was done on admission and repeated based upon the progression of the disease.

Obstetric ultrasound with fetal doppler were performed in patients on expectant management.In case of any abnormalities like Doppler changes, oligohydromnios and IUGR then expectant management was discontinued and planned for termination.

Details regarding treatment (Antihypertensives, Mgso4, steroids) were noted.

Details regarding mode of termination and indication for termination, Intraoperative,postpartum complications were noted.

Neonatal assessment was done by following the babies in the NICU.Maternal complications were noted. Follwup of mother and neonate done upto discharge / death.

Collected datas were tabulated and statistical analysis performed.

OBSERVATION AND RESULTS

TABLE: I

AGE DISTRIBUTION

AGE	NUMBER OF PATIENTS	PERCENTAGE
<20	33	22
21-30	90	60
>30	27	18
Total	**150**	**100**

Most of the women were in the age group 21 – 30 (60%).

Mean age group was 26 Yrs, with a range of 17 yrs - 40 yrs.

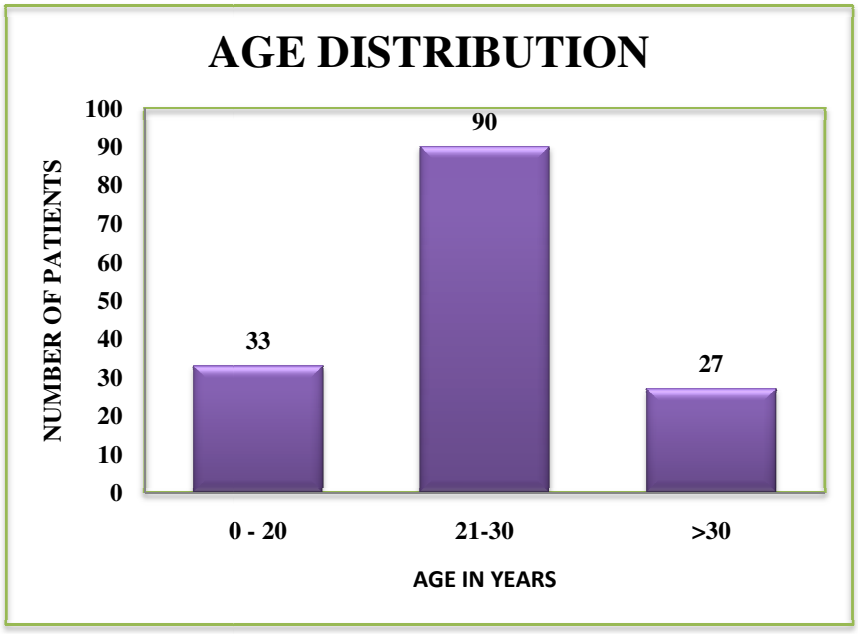


TABLE : II

PARITY

PARITY	NUMBER OF PATIENTS	PERCENTAGE
PRIMI	99	66
MULTI	51	34
Total	**150**	**100**

Most of the patients were primi, n=99 (66%)

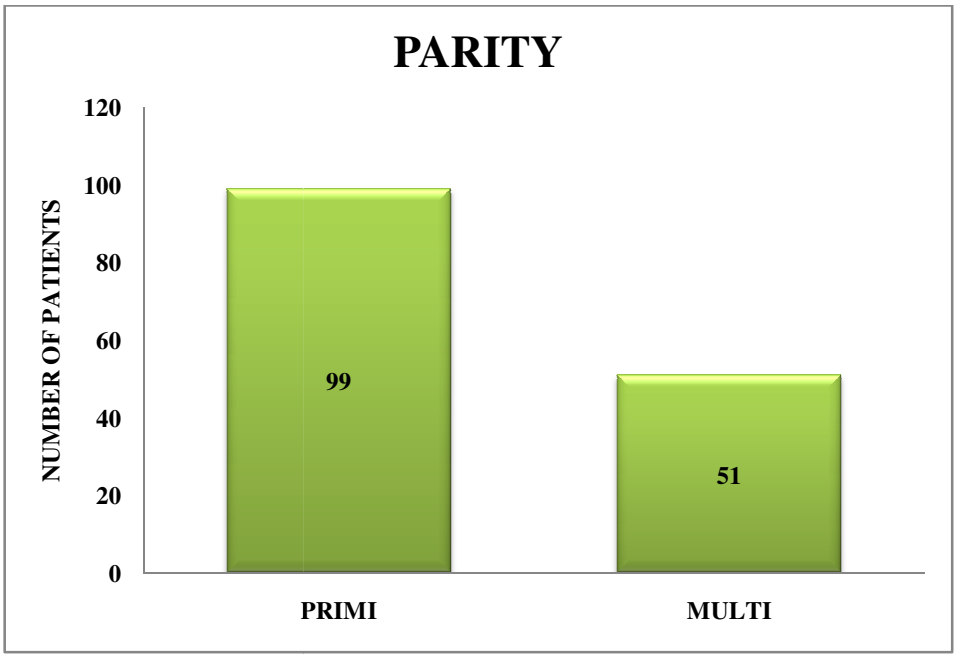


TABLE : III

ANTENATAL VISITS

ANTENATAL VISITS	NUMBER OF PATIENTS	PERCENTAGE
Booked	148	98.7
Unbooked	2	1.3
Total	**150**	**100**

Out of 150 patients 148 (98.6%) were booked and 2 (1.3%) were unbooked

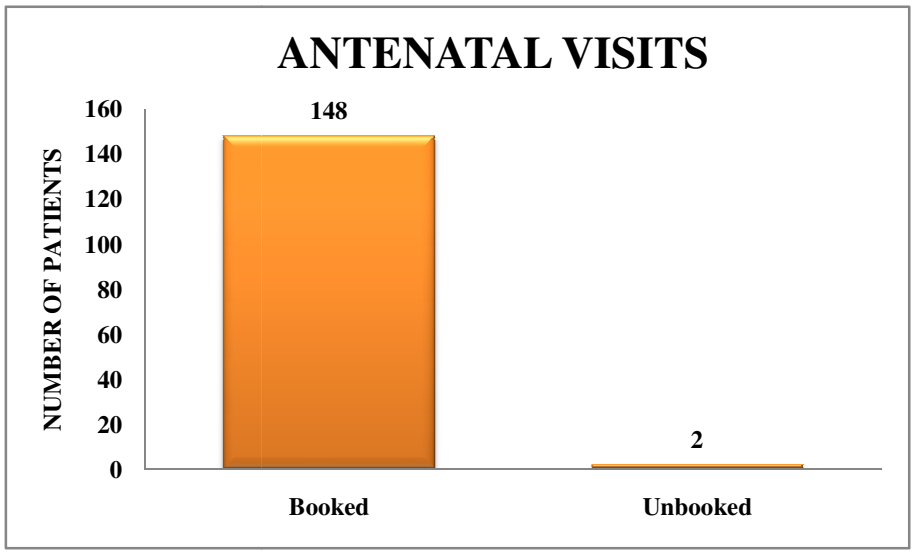


TABLE: IV

SOCIOECONOMIC CLASS

SOCIOECONOMIC CLASS	NUMBER OF PATIENTS	PERCENTAGE
I	…	…
II	…	…
III	1	0.7
IV	85	56.7
V	64	42.6
Total	**150**	**100**

Most of the women belonged to social economic class IV and V.

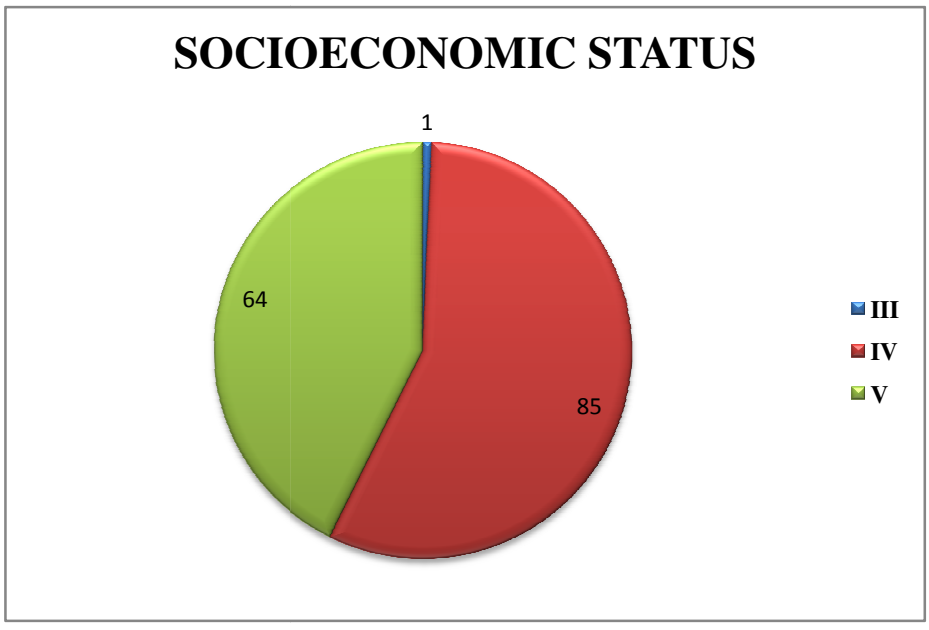


TABLE : V

GA AT DIAGNOSIS

GA AT DIAGNOSIS (WEEKS)	NUMBER OF PATIENTS	PERCENTAGE
24 – 28	21	14
28 – 32	53	35.3
32 – 34	76	50.7
Total	**150**	**100**

This table includes gestational age at which the patient first fulfilled the inclusion criteria for severe preeclampsia.

Most of women n = 76 (50.7%) were in 32 – 34 weeks Mean gestational age at diagnosis was 31 weeks with a range of 25 to 34 weeks.

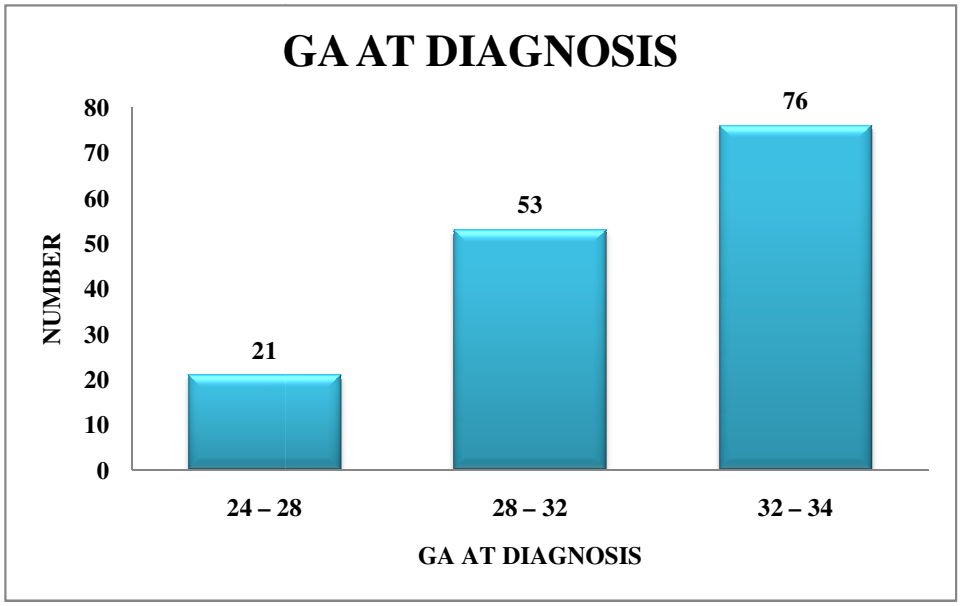


TABLE – VI

GA AT DELIVERY

GA AT DELIVERY	NUMBER OF PATIENTS	PERCENTAGE
24-28	13	8.7
28-32	51	34
32-34	84	56
> 34 WEEKS	2	1.3

Most of the Women underwent termination of pregnancy after 32 weeks 84(56%)

Mean gestational age at the time of delivery was 32 weeks.

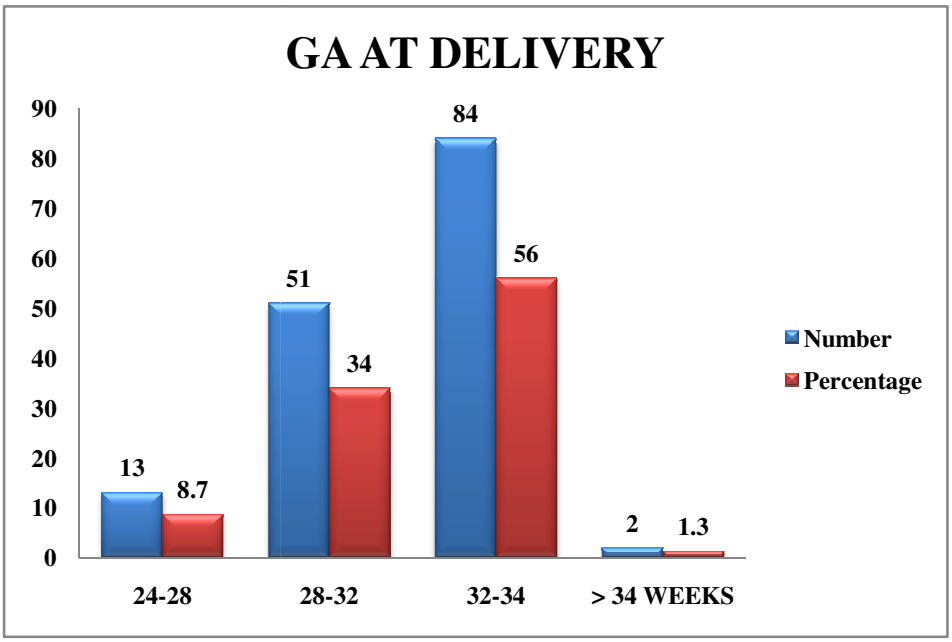


TABLE : VII

INVESTIGATIONS:-

INVESTIGATIONS	NUMBER OF PATIENTS	PERCENTAGE
Hyperuricemia	78	52
Altered LFT	30	20
Thrombocytopenia	23	15.3
Altered RFT	30	20
Fundus Change	33	22
DOPPLER Changes	12	8

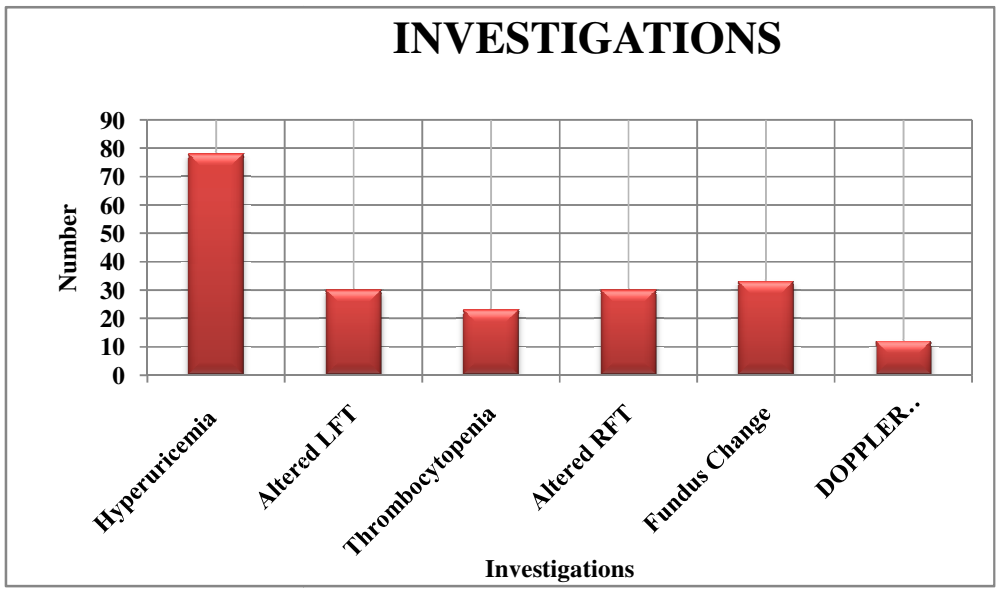


TABLE : VIII

MGS04 :

MGS04	NUMBER OF PATIENTS	PERCENTAGE
Received	104	69.3
Not Received	46	30.7

Out of 150 patients, 104 (69.3%) women received Mgs04, out of which 4 developed seizures 3 of them had Post partum eclampsia.

46 (30.7%) women did not received Mgs04, out of which 11 (23.4%) developed seizure. Mgs04 was given in view of imminent symptoms and signs. In 26 cases Mgs04 was given in view of high BP without imminent symptoms.

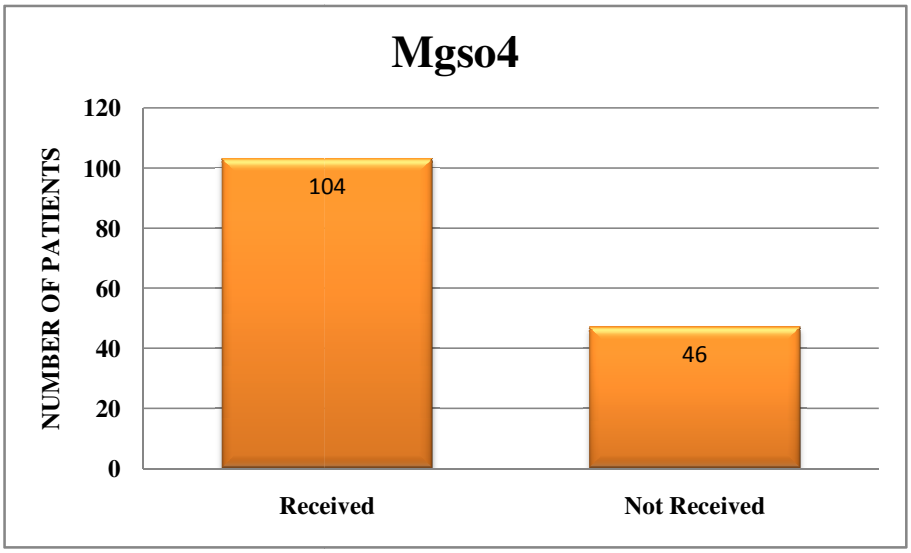


TABLE : IX

ANTI HYPERTENSIVES:-

ANTIHYPERTENSIVES	NUMBER OF PATIENTS	PERCENTAGE
Oral	150	100
Parenteral	33	22

All women in this study group received oral antihypertensive (methyldopa with Nifedipine or Labetalol with Nifedipine)

33 women (22%) required parenteral antihypertensive (Labetalol or Nitroglycerine) for control of blood pressure.

Out of 150 women, 72 (48%) women were on oral antihypertensive before admission.

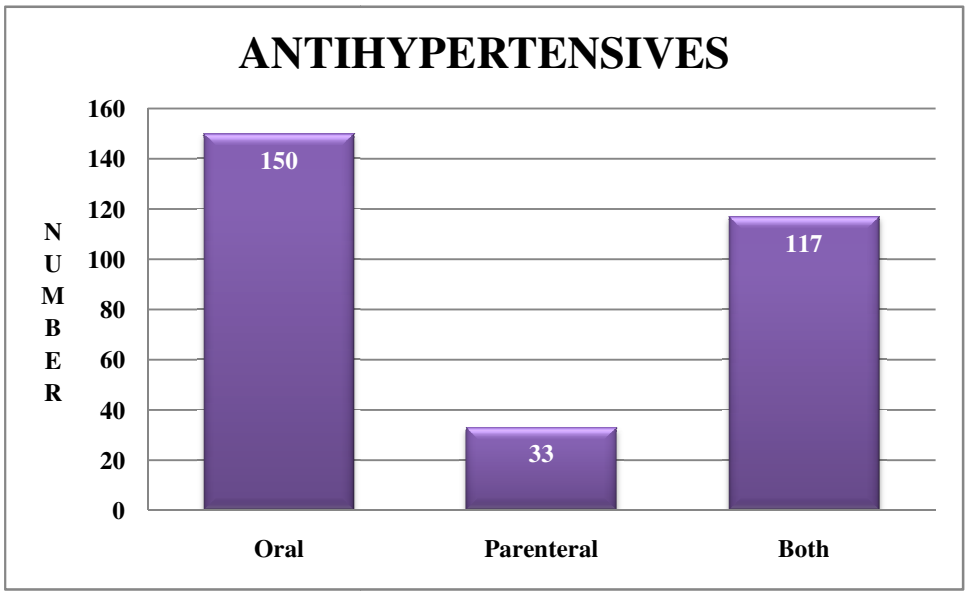
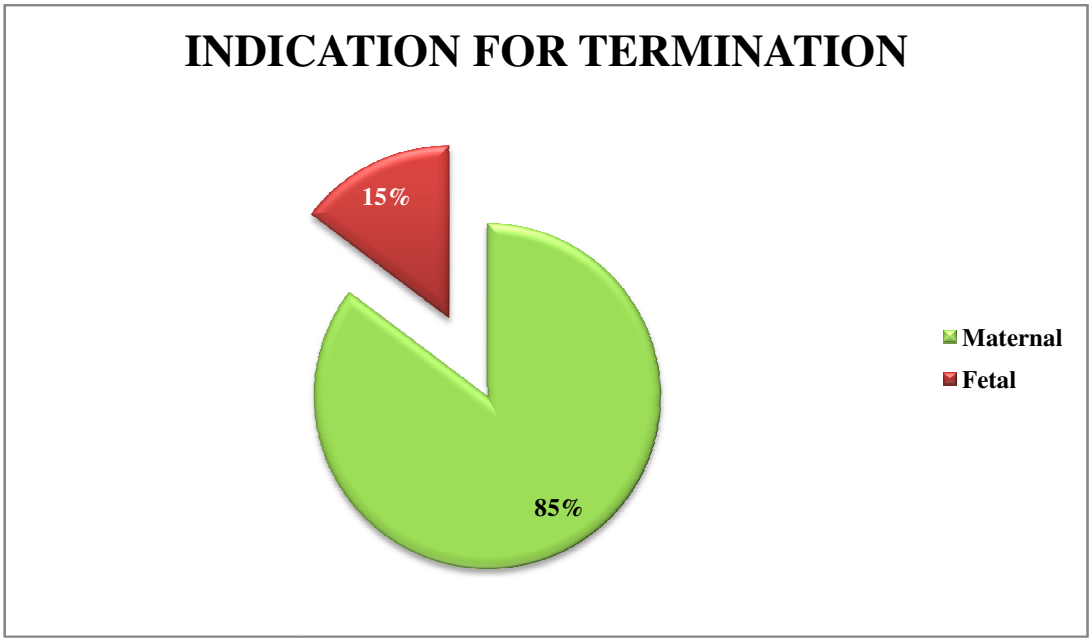


TABLE – X

INDICATION FOR TERMINATION OF PREGNANCY

INDICATION	NUMBER OF PATIENTS	PERCENTAGE
Maternal	128	85.3
Fetal	22	14.7

Maternal indication was found to be the most common cause for termination of pregnancy, 128 (85.3%) most common cause being imminent eclampsia. Most common fetal indication was Doppler changes.



INDICATIONS FOR TERMINATION ARE THE FOLLOWING:

MATERNAL

1. Signs of imminent eclampsia
2. Eclampisa
3. Uncontrolled hypertension in spite of oral and parenteral anti-hypertensive
4. Abruptio placenta
5. Abnormalities in renal function test
6. HELLP Syndrome
7. DIVC

FETAL

1. Doppler abnormalities
2. Severe oligohydromnios
3. IUGR

TABLE – XII

MODE OF DELIVERY

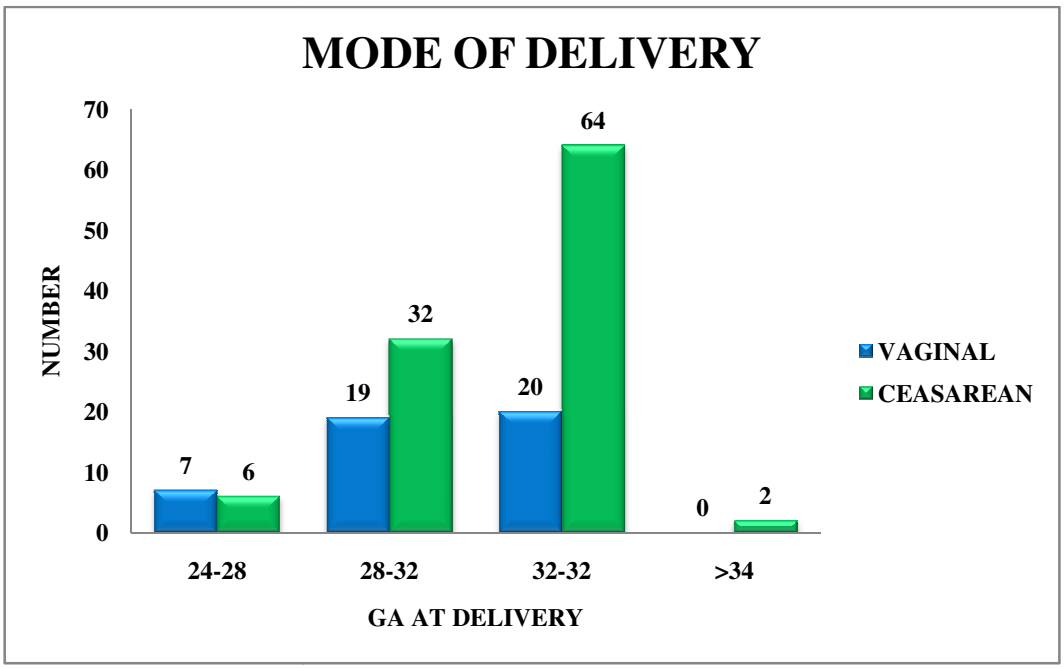
GA AT DELIVERY	VAGINAL	CAESAREAN	TOTAL
24-28	7	6	13
28-32	19	32	51
32-34	20	64	84
>34	0	2	2
TOTAL	**46**	**104**	**150**

Out of 150 patients, 69 (46%) were induced.

Out of 69 patients who were induced , 46 (30.7 %) delivered vaginally and 23 (15.3%) underwent caesarean section.Out of 25 IUFDs, 9 were delivered by caesarean section indication being Abruption for 7 and eclampsia for 2 patients.

81(54%) patients were not induced and underwent direct caesarean section. The causes were Imminent eclampsia, eclampsia , Abruption, Doppler changes, severe oligohydromnios.

Overall , total number of patients who underwent LSCS were ,104(69 %)


MODE OF DELIVERY
70
60
50
40
30
20
10
0
NUMBER
7
6
19
32
20
64
0
2
24-28
28-32
32-32
>34
GA AT DELIVERY
VAGINAL
CEASAREAN

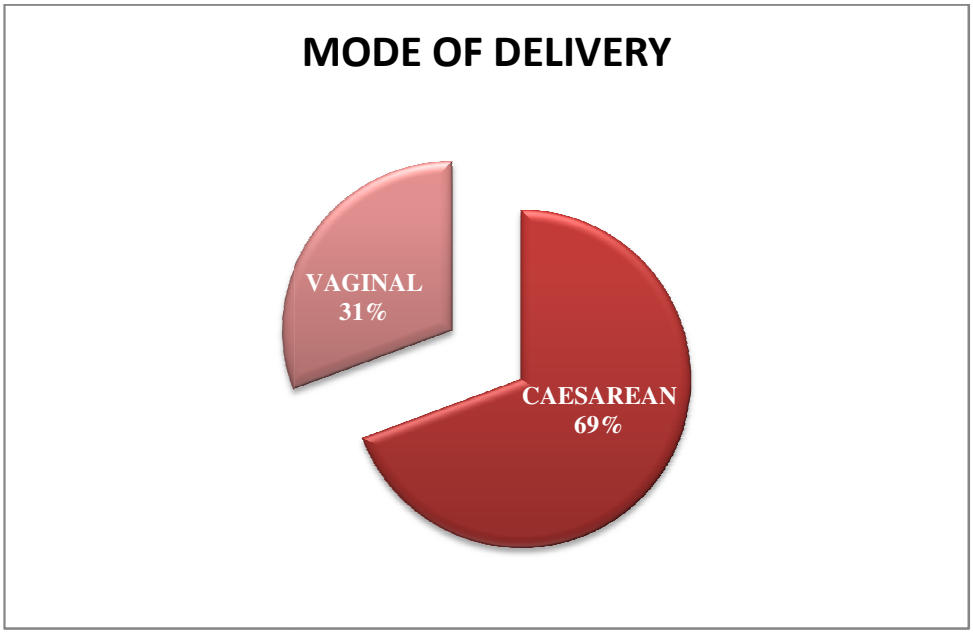
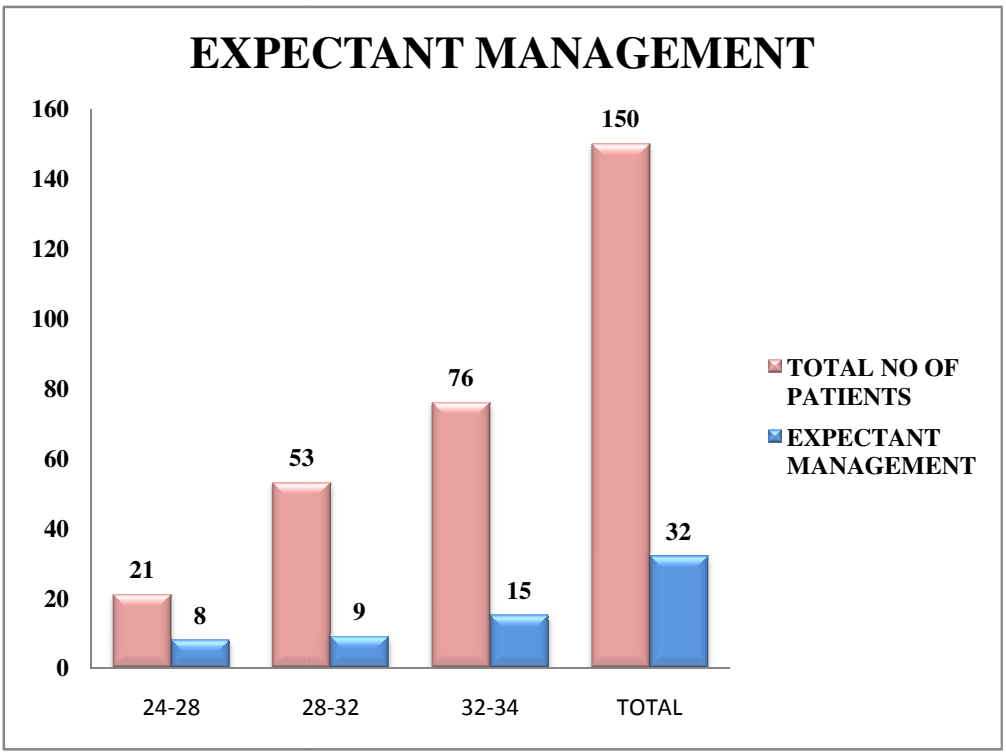

MODE OF DELIVERY
VAGINAL
31%
CAESAREAN
69%

TABLE – XII

EXPECTANT MANAGEMENT

GA AT DIAGNOSIS	NUMBER OF PATIENTS	EXPECTANT MANAGEMENT	
		NUMBER OF PATIENTS	PERCENTAGE
24-28	21	8	38.1
28-32	53	9	16.9
32-34	76	15	19.7
TOTAL	150	32	21.3

Out of 150 patients in this study 32(21.3%) patients underwent expectant management. Maximum prolongation of pregnancy was 25 days.


EXPECTANT MANAGEMENT
160
140
120
100
80
60
40
20
0
21
8
53
9
76
15
150
32
24-28
28-32
32-34
TOTAL
TOTAL NO OF PATIENTS
EXPECTANT MANAGEMENT

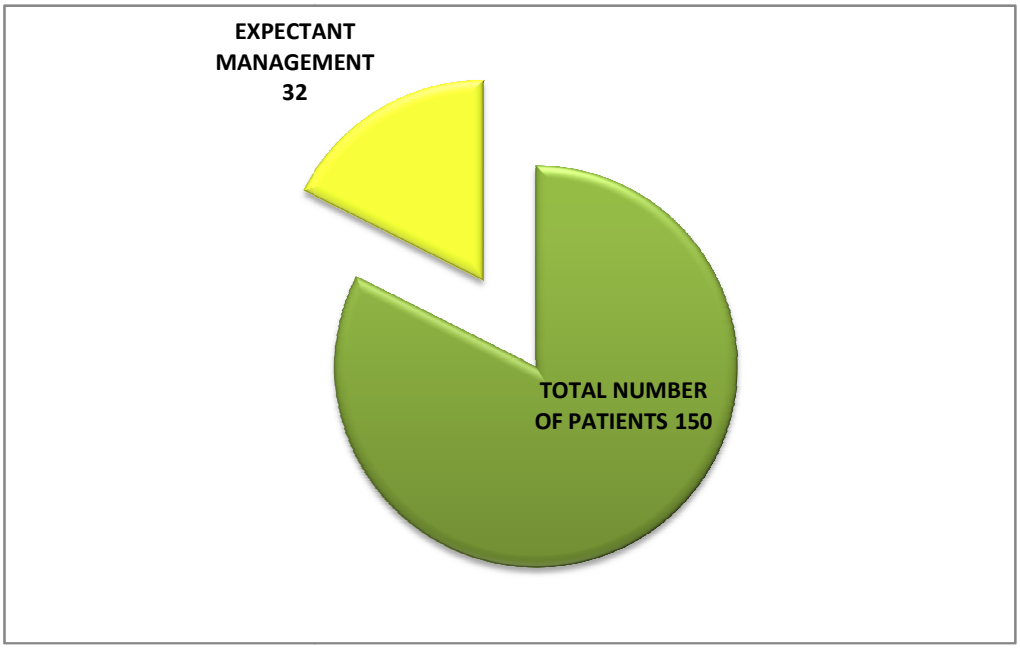

EXPECTANT MANAGEMENT 32
TOTAL NUMBER OF PATIENTS 150

TABLE – XIII

PROLONGATION OF PREGNANCY

GA AT DIAGNOSIS	NO OF DAYS GAINED			TOTAL
	1-10	11-20	>20	
24-28	6	2		8
28-32	7	1	1	9
32-34	14	0	1	15
TOTAL	27	3	2	32

Out of 32 patients, 27(87.4%) patients had a prolongation of pregnancy ≤ 10 days. Maximum prolongation was 25 days. Mean number of days gained was 7 days with a range of 1 – 25 days.

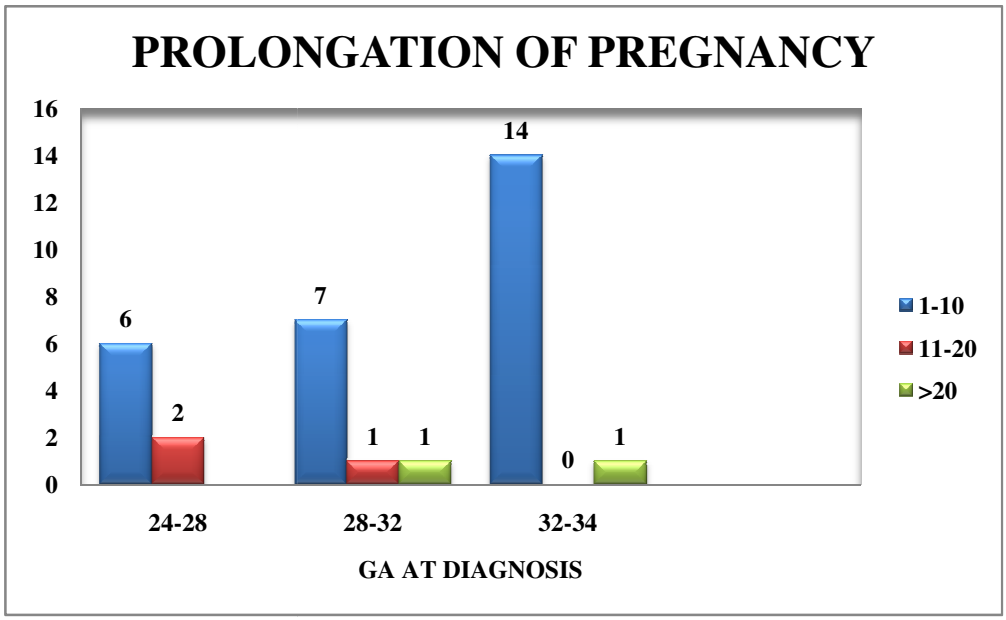


TABLE : XIV

GA AT DIAGNOSIS Vs OUTCOME:

GA at Diagnosis (Weeks)	Number of Patients	Maternal Morbidity and Mortality		Fetal Morbidity and Mortality	
		No	%	No	%
24 – 28	21	11	52.3	20	95.2
28 – 32	53	20	37.7	37	69.8
32 – 34	76	25	32.8	33	43.4
Total	150	58	38.6	90	60

Both maternal and fetal morbidly and mortality were more during early gestational age, 52.3%and 95.2% at gestational age 24 – 28 weeks respectively. Overall fetal morbidity was more than maternal morbidity.

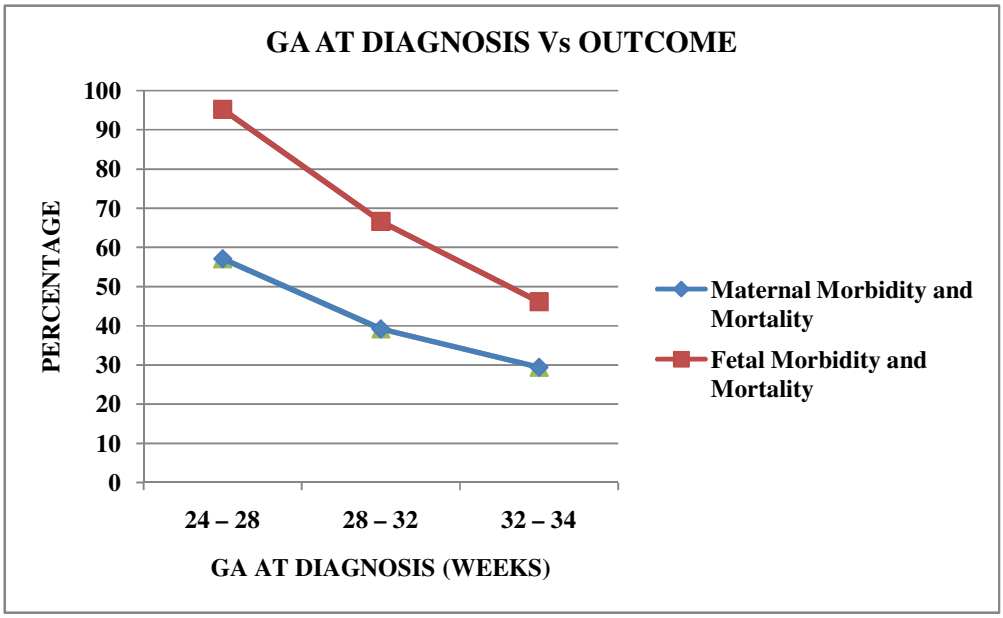


TABLE – XV

GA AT DELIVERY (VS) FETAL MORBIDITY AND MORTALITY(Excluding 25 IUFDS)

GA AT DELIVERY (Weeks)	RDS		HIE		IUGR		PND	
	NO	%	NO	%	NO	%	NO	%
24-28 (13)	4	30.7	1	7.69	1	7.69	5	38.4
28-32 (51)	7	13.7	5	9.8	6	11.7	10	19.6
32-34 (84)	11	13	4	4.76	9	10.7	5	6
>34 (2)	-		-		-		-	

Fetal morbidity and mortality was found to be high at early gestational age,38.4% perinatal death at 24 – 28 weeks where as it is 6% at 32- 34 weeks.RDS was high between 24-28 and 28-32 weeks,30.7% and 13.7% respectively. **P value < 0.05, statistically significant.**There was positive correlation between geastational age at delivery and fetal morbidity and mortality.

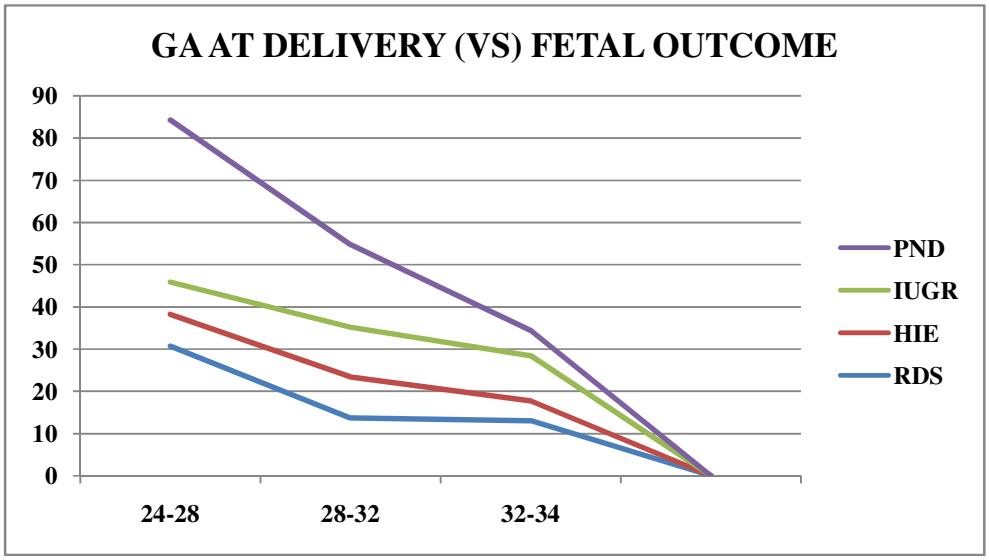


TABLE – XVI MATERNAL OUTCOME

MATERNAL OUTCOME	NUMBER OF PATIENTS	PERCENTAGE
Eclampsia	17	11.3
Abruption	14	9.3
Wound Infection	8	5.3
DEATH	**6**	**4**
Post partum eclampsia	3	2
HELLP	3	2
DIVC	3	2
ARF	2	1.3
Atonic PPH	2	1.3
TOTAL	58	38.6

58 (38.6%) women experienced complications. Eclampsia and abruption were highest accounting to 17(11.3%) and 14(9.3%) respectively.There were 6 (4%) maternal death in this study.All the six maternal death were between the gestational age of 30 to 34 weeks.

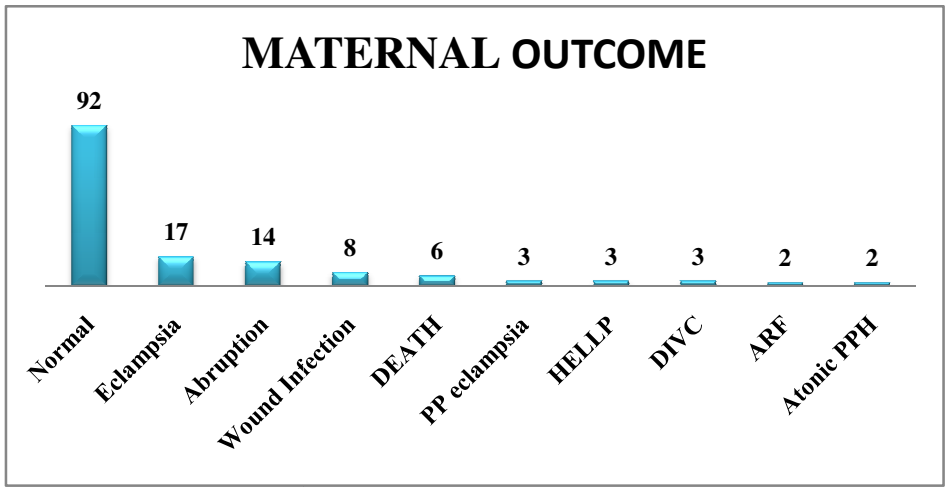


TABLE – XVII

MATERNAL DEATH

CAUSES OF MATERNAL DEATH	NUMBER OF PATIENTS
HELLP / DIVC	**2**
AP Eclampsia with hypertensive failure	**1**
AP Eclampsia with ARF	**1**
Severe preeclampsia with pulmonary edema	**1**
Abruption with DIVC	**1**
TOTAL	**6**

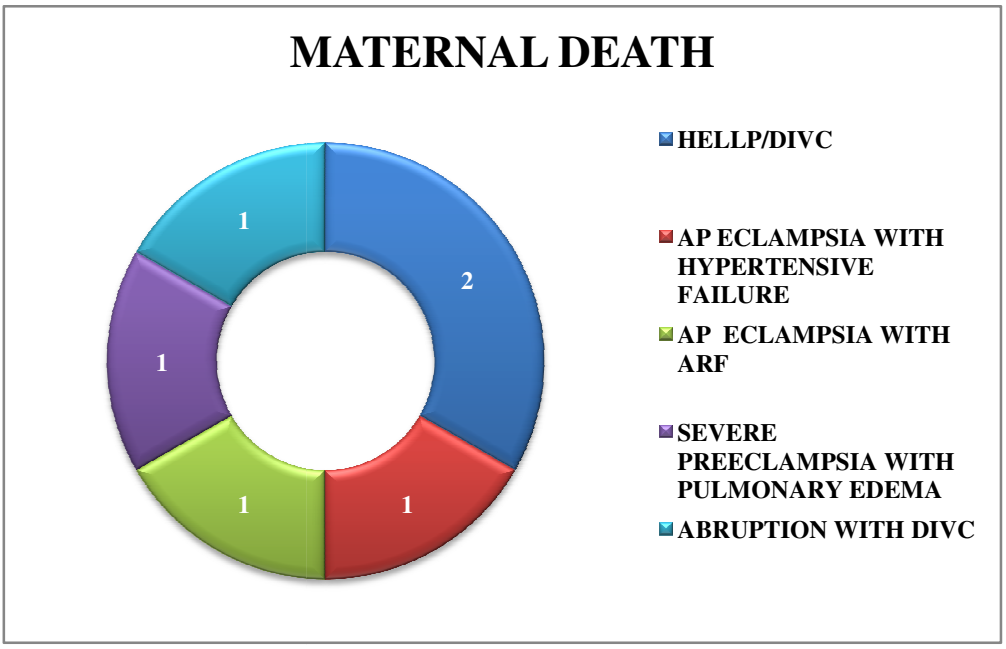


TABLE – XVIII

FETAL OUTCOME

FETAL OUTCOME	NUMBER OF BABIES	PERCENTAGE
IUFD	25	27.8
IUGR	15	16.6
Death	16	17.8
RDS	13	14.4
HIE	2	1.3
RDS +HIE	9	10
SEPTICEMIA	6	6.7
STILLBORN	4	4.5
TOTAL	**90**	**100**

Out of the total 150 babies , 90(60%) babies had complication. Out of 90 babies 25(16.6%) were IUFDs,majority were in women who were referred as IUFDs.Cause of IUFD being abruption in majority of cases.121 babies were born alive. All were preterm. Major cause for neonatal morbidity and mortality were prematurity and Respiratory distress syndrome.Out of 21 babies who had RDS, 13(62%) did not receive steroids and 8(38%) received steroids. Neonatal ICU admission were needed in 90% of babies. Neonatal deaths were 16(10.6%)

PN Mortality and Morbidity

Total no of babies → 150

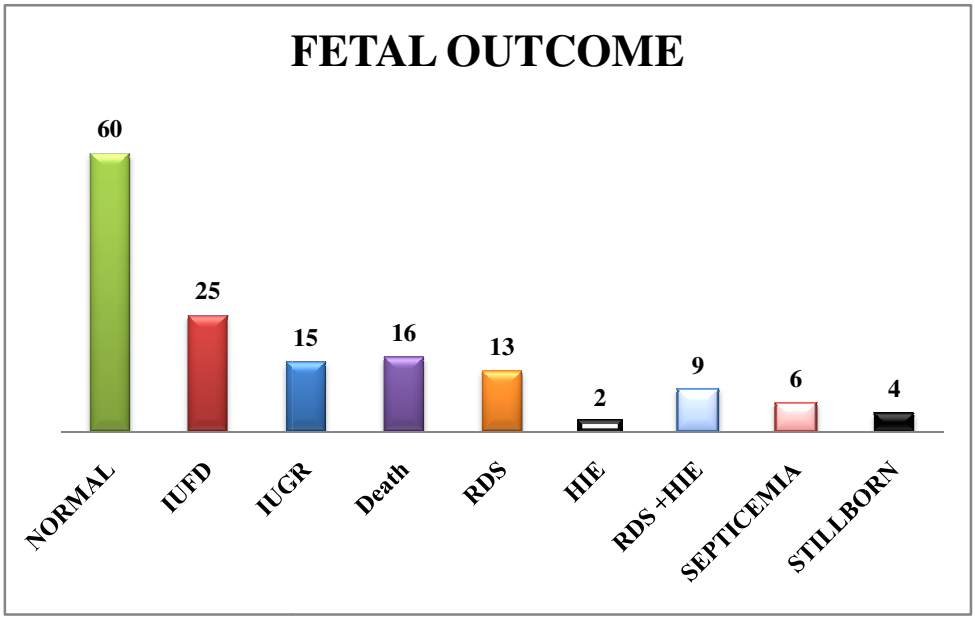
No of live babies → 121

No of IUFDs → 25

No of Stillbirth → 4

No of Neonatal death → 16

Total Perinatal death →IUFD+SB+ND →45
Incidence of PNM →Total PND /No of babiesX 100 →30%



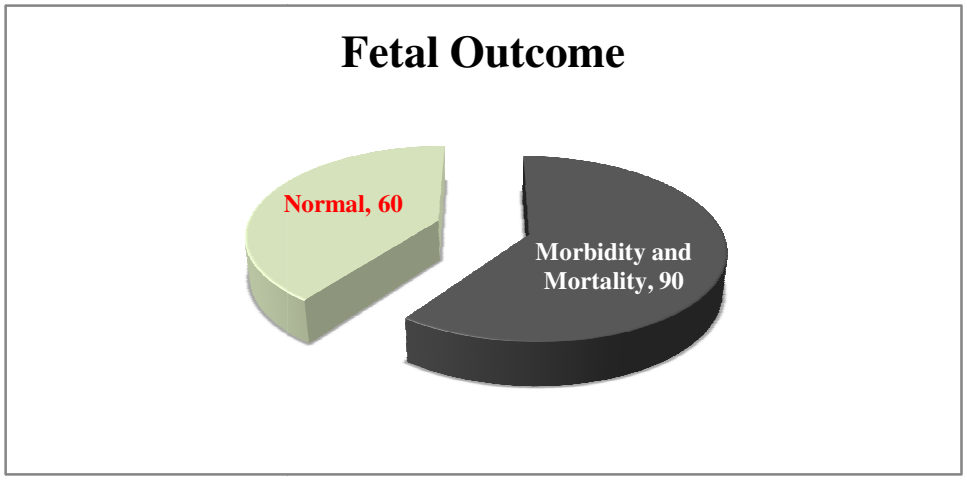


TABLE – XIX

GA AT DELIVERY Vs 5 MIN APGAR

GA AT DELIVERY	APGAR			
	0	1-4	5-7	8-10
24-28	7	2	4	0
28-32	16	3	23	9
32-34	7	6	43	28
>34	0	0	1	1
TOTAL	30	11	71	38

Out of 121 babies (excluding 25 IUFDs and 4 stillborn), 71 (47.3%) babies had apgar of 5-7. Apgar was found to be improving with increasing gestational age, which was statistically significant with P value <0.002.

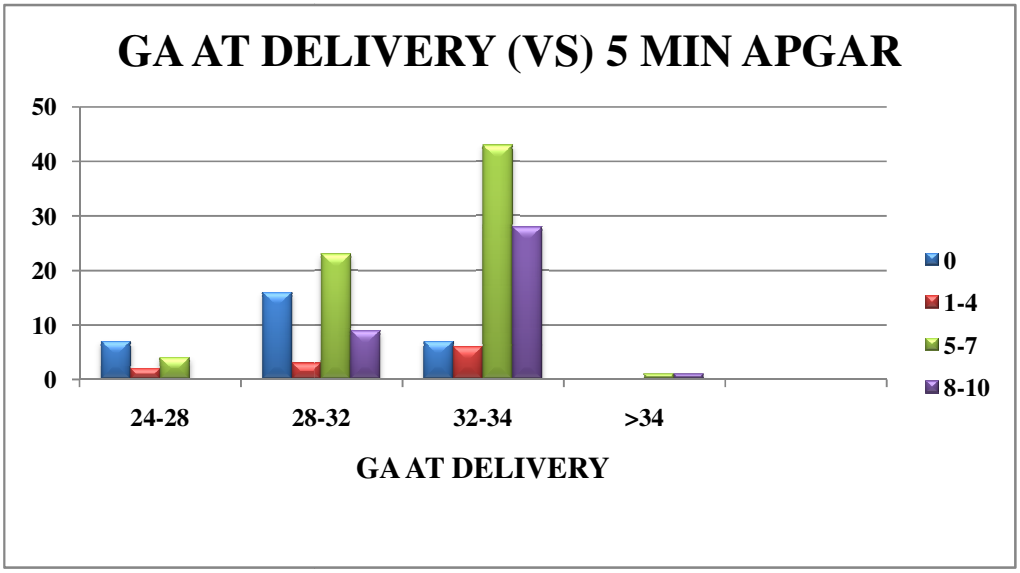


TABLE – XX

GA AT DELIVERY Vs BIRTH WEIGHT

GA AT DELIVERY	BIRTH WEIGHT IN GRAMS			
	500-999	1000-1499	1500-1999	2000-2499
24-28	9	4	-	-
28-32	7	33	9	2
32-34	1	16	54	13
>34	-	-	2	-
TOTAL	17	53	65	15

Low birth weight, defined as 1.5-2.5kg was seen in 65 (43.3%) cases very low birth weight 1-1.5Kg was seen in 53 (35.3%) extreme low birth weight <1kg was seen in 17 (11.3%) cases. Mean birth weight was 1.43kg with a range of 500 grms to 2300 grms. IUGR was seen in 23(19.1%) babies.

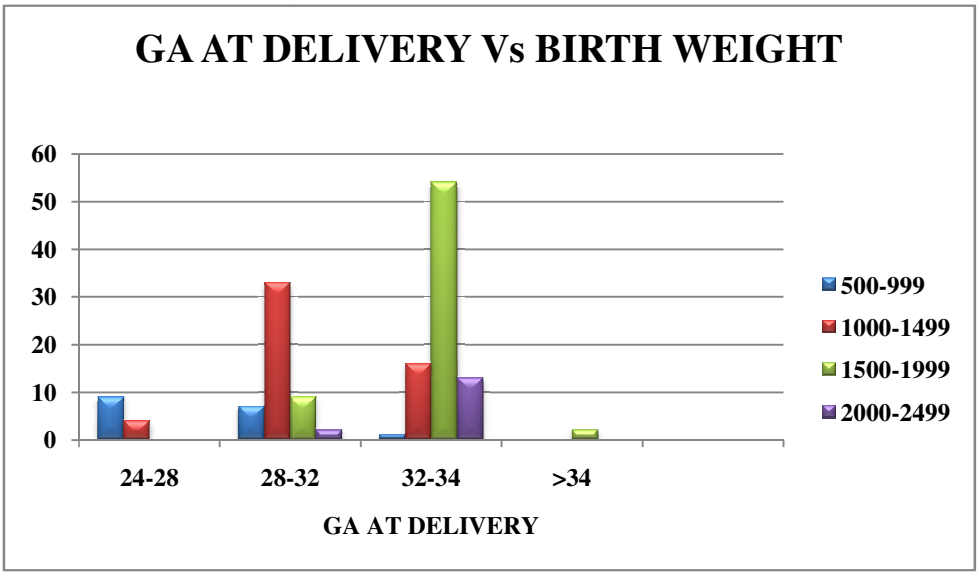


TABLE – XXI
BIRTH WEIGHT (VS) FETAL OUTCOME (Excluding 25 IUFDS)

BIRTH WEIGHT (Grams)	RDS		PND		ALIVE	
	NO	%	NO	%	NO	%
500-999			7	100		
1000-1499	15	35.7	10	23.8	32	76.1
1500-1999	10	16.3	3	4.9	58	95
>2000	-		-		14	93.3

There is rapid fall in death rate and perinatal morbidity as birth weight increases.

PND was 100% in birth weight <1 kg where as there was no perinatal death in birth weight >2 kg. **p value is<0.001 statistically significant.**There was a positive correlation between birth weight and outcome

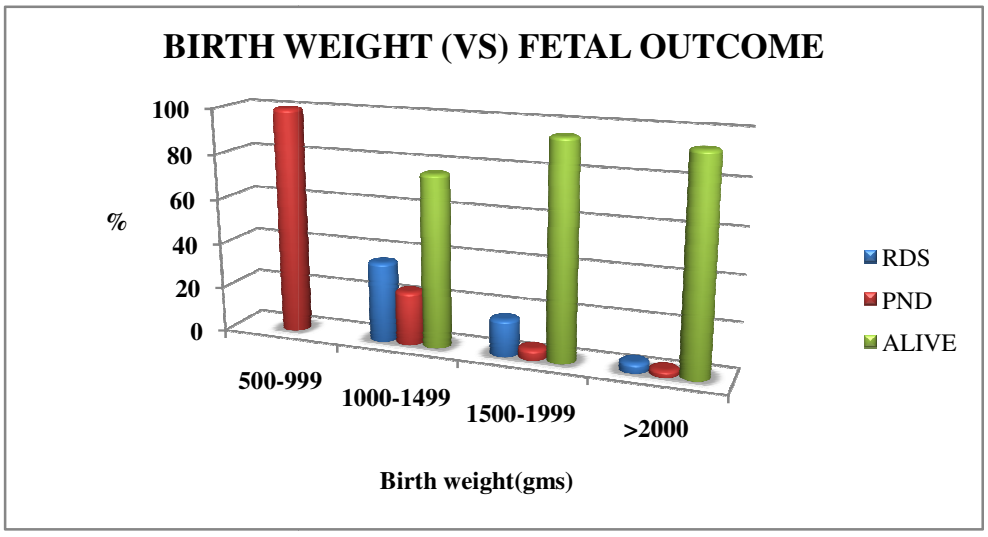


TABLE – XXII

EXPECTANT MANAGEMENT (VS) FETAL OUTCOME

PROLONGATION OF PREGNANCY (DAYS)	FETAL MORBIDITY		FETAL MORTALITY	
	NO	%	NO	%
1-10 (27)	7	25.9	1	3.7
11-20 (3)	2	66.9	1	33.3
>20 (2)	-	-	-	-
TOTAL (32)	9	28.1	2	6.2

Out of 32 patients who had expectant management there was 9(28.1%) and 2(6.2%) fetal morbidity and mortality respectively.There were no still birth.

Two neonatal death occurred in patient who were delivered at 29 weeks.

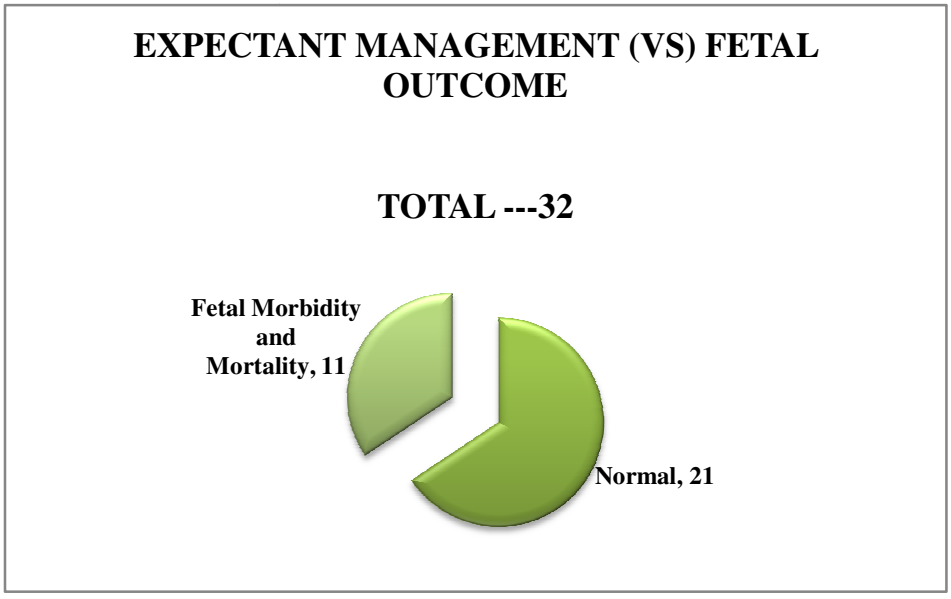


TABLE – XXIII

EXPECTANT MANAGEMENT (VS) MATERNAL OUTCOME

MATERNAL COMPLICATIONS	NUMBER OF PATIENTS
Eclampsia	1
HELLP	1
ARF	1
PP Eclampsia	1

Out of 32 patients who had expectant management there were no maternal death, and 4(12.5%) patients had complications.All patients were delivered by caesarean section.

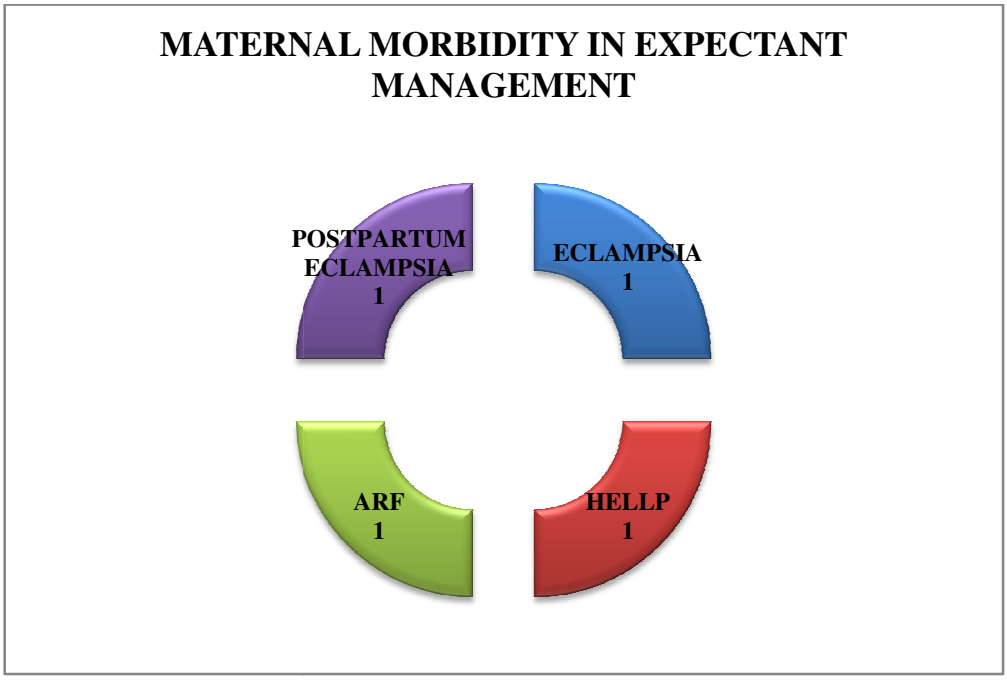


TABLE – XXIV
POST OP BP CONTROL

BP CONTROL	NUMBER OF PATIENTS	PERCENTAGE
<3 DAYS	18	12.5
3-7	107	74.3
8-14	16	11.1
>14	3	2.1

In majority of the patients BP control was achieved within a week.

only 7 patients were discharged with antihypertensives.

TABLE – XXV
POSTPARTUM HOSPITALISATION

NO OF DAYS	NUMBER OF PATIENTS	PERCENTAGE
<7	5	3.5
7-14	69	45.8
15-28	69	47.9
>28	4	2.8

Most of the women required hospitalisation for 2 to 3 weeks with a minimum of 7 days and a maximum of 31 days.

Mean of postpartum hospital stay was 16 days.

Prolonged hospitalisation in most of the women was for baby sake

DISCUSSION

Preeclampsia affects both mother and neonate. It is one of the leading cause of maternal and fetal morbidity and mortality.

AGE:

Sixty (60%) of the women in the study group were in the age group of 21 to 30 years, which correlates with the studies of **Moodley**[83] in which the mean age was 26 years.In studies done by **Brown MA and Buddle ML**[33]**, D.R.Hall**[14] the mean age was 26 years.In our study mean age was 26 years.

PARITY:

Preeclampsia is common in first pregnancy.More than half the women in this study were Nulliparous, 99 (66%). **Brown MA and Buddle ML**[33] said preeclampsia is predominant in nulliparous.

RISK FACTORS:

Only forty three (28.6%) of the women in this study group had risk factors, which includes gestational hypertension in previous pregnanacy and family history of hypertension.

In the study by **D.R.Hall**[14] 36% of the women had risk factors.

ANTENATAL VISITS:

Almost one forty eight (98.6%) women in this study were booked either at our institution or outside. 108 (68%) were booked outside and referred to IOG.

Adequate antenatal care has an important role in reducing the complications by early detection and appropriate management. Though most of the patients in this study were booked , early detection of gestational hypertension was not made because one or two recordings of high blood pressure at early gestational age was not taken into serious consideration and also because of unawareness of the women.

SOCIOECONOMIC STATUS:

Most of the women , 85 (56.7%) and 64(42.6%) belonged tosocioeconomic class IV and V respectively.

GA AT DIAGNOSIS:

Mean gestational age at diagnosis was 31 weeks in our study.

AUTHOR	GA AT DIAGNOSIS(WEEKS)
D.R.Hall	**30**
Brown MA and Buddle ML	27
J.Moodley and Daya	32
Our study	**31**

There was a positive correlation between gestational age at diagnosis and the Fetal outcome.

Earlier the onset of preeclampsia greater is the fetal complications, 95.2% at 24 to 28 weeks compared to 43.4% after 32 weeks.

INVESTIGATIONS:

Serum uric acid was found to be high in 52% of the patients.Uric acid is used as an indicator of disease severity and a good predictor in determining the outcome of the fetus than blood pressure[21].

Platelet count less than 100,000mm^3 is considered as an indicator of severity of preeclampsia[31,85].In this study 15.3% of the

women had a low platelet count.Platelet count is decreased because of increase in consumption and destruction inside the vessel lumen[86.]

Coagulation profile was altered in 11(7.3%) and 30 (20%) patients had altered renal functions.Fundal changes of grade I and grade II were found in 33 (22%) of patients in which Grade I and Grade II were 22(14.7%) and 11(7.3) respectively.There were no ophthalmic indications like papilledema and retinal detachment for termination of pregnancy.

MAGNESIUM SULPHATE :

Magnesium sulphate reduces the risk of maternal mortality and the risk of seizures[88.] Magnesium sulphate was given to 69.3% of severe preeclampsia in our study which is low compared to other studies[88,89.]In a study by **Lee WO' Connell CM and Baskett** 97% of the patients received magnesium sulphate [89.]

Magnesium sulphate when given to patients with severe preeclampsia reduces the risk of seizures by 58%.But routine prophylaxis in patients with severe preeclampsia is questioned and the decision is based on the method of treatment whether conservative or immediate termination.

ANTIHYPERTENSIVE AGENTS:

In this study all patients received oral hypertensives either Methyldopa and nifedipine or labetolol and nifedipine. 33 (22%) patients required parenteral antihypertensives in addition to oral antihypertensive.

In all patients who required parenteral antihypertensive magnesium sulphate was given and seizures didnot occur.

GESTATIONAL AGE AT DELIVERY:

Most of the women underwent termination of pregnancy at 32-34 weeks , 84(56%).In the study by **D.R.Hall**[14] GA at the time of delivery was found to be 32-34 weeks.Apgar was found to be improving with increasing gestational age.

Fetal morbidity and mortality was found to be high at early gestational age,38.4% perinatal death at 24 – 28 weeks where as it is 6% at 32- 34 weeks.RDS was high between 24-28 and 28-32 weeks,30.7% and 13.7% respectively.

Dehram et al[34] showed improvement in neonatal survival with increasing gestational age.

TERMINATION OF PREGNANCY:

Maternal indication was found to be the most common cause for termination of pregnancy in 128(85.3%) patients and the fetal cause in 22(14.7%).Imminent eclampsia was the most common cause of maternal indication for termination..In the study by **Blackwell SC in 2002**, termination was done for maternal indication in 80% and for fetal indication in 20% of the patients.

Though preeclampsia is one of the risk factor for prematurity, the cause of prematurity in most of the cases is iatrogenic either induction of labour or caesarean section to prevent morbidity and mortality in mother and fetus

MODE OF DELIVERY:

Though delivery is the ultimate cure for preeclampsia, fetal outcome should be taken into consideration in the absence of maternal complication[21,86].

Almost 69% of the women in this study were delivered by caesarean section.

This rate is higher than that reported by **Mashiloane and Moodley[83]** but similar to that of Hall et al where 81.5% delivered by means of caesarean section[14].

STUDY	VAGINAL DELIVERY	CAESAREAN
Hall et al	18.5%	81.5%
Murphy DJ	20%	80%
Nassar et al	48.3%	51.7%
Railton and Allen	25%	75%
Our study	**31%**	**69%**

Our study correlates with that of Railton less than that of Hall and Murphy and higher than Nassar.

FETAL PARAMETERS:

APGAR:

Out of 120 babies (excluding 25 IUFDs and 4 stillborn), 71 (47.3%) babies had apgar of 5-7.

Apgar was found to be improving with increasing gestational age.Only 5.6% babies had apgar of 5-7 at 24 to 28 weeks.

BIRTH WEIGHT:

Low birth weight, defined as 1.5-2.5kg was seen in 65 (43.3%) cases very low birth weight 1-1.5Kg was seen in 53 (35.3%) extreme low birth weight <1kg was seen in 17 (11.3%) cases. Mean birth weight was 1.43kg.

The mean birth weight was 1.4 kg in the study by **D R Hall et al**[14] , 1.62 kg in the study by **Sibai et al.**

There is rapid fall in death rate and perinatal morbidity as birth weight increases. PND was 100% in birth weight <1 kg where as there was no perinatal death in birth weight >2 kg.

Odendal et al[90] showed improving perinatal survival as birth rate increases.

MATERNAL OUTCOME:

Ultimate goal in the management of severe preeclampsia must first be the safety of the mother and second the delivery of a live infant who will not require prolonged neonatal care.

In our study maternal morbidity and mortality was seen in 38.6%. Eclampsia and abruption were highest accounting to 17(11.3%) and 14(9.3%) respectively.Other complications were HELLP,DIVC,ARF.

In our study Maternal mortality was 6 (4%) . In study conducted by Manisha et al in New delhi in 2012 reported maternal mortality of 1.8% in their study .

STUDY	ABRUPTION	PULMONARY EDEMA	HELLP	ECLAMPSIA	RENAL FAILURE
DR Hall	20%	2%	5%	1.2%	0.3%
Vissur and Wellenberg	5%	-	-	1.9%	-
Murphy DJ	1.5%	-	21%	1.4%	1.3%
Olah and edman	-	-	14.2%	-	3.5%
Manish et al	1.9%	1.9%	10.4%	12.8%	-
Our study	**10%**	**1.3%**	**3.3%**	**14.6%**	**2%**

PERINATAL OUTCOME:

Total Perinatal death →IUFD+SB+ND →45.

Incidence of PNM →Total PND / No of babies X 100 →30% .

The neonatal outcome depends on the intensive care facilities and the gestational age at birth.**Witlin et al**[94] reported that neonatal outcome in early onset severe preeclampsia was directly correlating with increasing birth weight and Respiratory distress syndrome reduced with increasing gestational age.

In our study 90% babies required neonatal ICU.Major neonatal complications were Hyaline membrane disease, HIE,IUGR, Septicemia,and neonatal death.

The administration of antenatal steroid has shown significant reduction in neonatal morbidity and mortality. Out of 21 babies who

had RDS, 13(62%) did not receive steroids and 8(38%) received steroids

PROLONGATION OF PREGNANCY (VS) OUTCOME:

Visser and Wallenburg[97] in their study of 254 women with severe preeclampsia between 20 to 32 weeks reported mean prolongation of pregnancy was 14 days and perinatal mortality was 20%.

Hall et al[14] reported in their study mean prolongation of pregnancy was 11 days and perinatal mortality was 24%. **Odendaal et al**[90] reported mean prolongation of pregnancy was 7.1 days.

In our study Out of 32 patients who had prolongation of pregnancy, 27(87.4%) patients had a prolongation of pregnancy ≤ 10 days. Maximum prolongation was 25 days. Mean Number of days gained was 7 days with a range of 1 – 25 days. There was 9(28.1%) and 2(6.2%) fetal morbidity and mortality respectively. There were no still births.Two neonatal deaths occurred in patients who were delivered at 29 weeks.

There were no maternal death in the expectant group of patients and 4(12.5%) patients had complications like Eclampsia, HELLP, ARF, PP Eclampsia.

POST OP BP CONTROL:

In majority of the patients BP control was achieved within a week. Only 7 patients were discharged with antihypertensives.

POSTPARTUM HOSPITALISATION:

Most of the women required hospitalisation for 2 to 3 weeks with a minimum of 7 days and a maximum of 31 days.Mean of postpartum hospital stay was 16 days.

Prolonged hospitalisation in most of the women was for baby sake. In the study by **D.R.Hall**[14] mean period of postpartum hospitalisation was 5 days.

SUMMARY

- In our study there were 150 patients of early onset severe preeclampsia.
- Most of them were in the age group of 21 – 30 years.
- Most of them were Primi (66%)
- Most of the women ,148 (98.6%) were booked.
- Most of the women , 85 (56.7%) and 64(42.6%) belonged to socioeconomic class IV and V respectively.
- Mean gestational age at diagnosis was 31 weeks.
- Only forty three (28.6%) of the women had risk factors (gestational hypertension in previous pregnanacy and family history of hypertension).
- Most patients had normal fundus.
- There was a significant reduction in the rate of seizures who received magnesium sulphate as prophylaxis.
- All patients received multidrug oral antihypertensives and 33 women received parenteral antihypertensive in addition to oral.
- Most of the women underwent termination after 32 weeks 84(56%)

- 85.3% termination was done for maternal indication,14.7% for fetal indication. Most common cause being Imminent Eclampsia.
- Though mode of delivery had no influence on fetal outcome, caesarean rate was higher which accounts to about 69%
- 32 patients was treated under expectant management and the maximum prolongation was 25 days.Mean number of days was 7 with a range of 1-25 days.
- Majority of babies had apgar between 5 and 7
- The mean birth weight is 1.43 kg.
- 58(38.6%) women experienced complications. Eclampsia and abruption were highest accounting to 17(11.3%) and 14(9.3%)
- There were 6(4%) maternal death. The cause of maternal death were HELLP / DIVC (2), AP Eclampsia with hypertensive failure (1), AP Eclampsia with ARF(1) , pulmonary edema(1) , Abruption with DIVC(1).
- 25(16.6%) were IUFDs, majority were in women who were referred as IUFDs
- Total Perinatal death →IUFD+SB+ND →45
- Incidence of PNM →Total PND /No of babiesX 100 →30%

- Major cause for neonatal morbidity and mortality were prematurity and Respiratory distress syndrome.
- Out of 32 patients who had expectant management there was 9(28.1%) and 2(6.2%) fetal morbidity and mortality respectively. There were no maternal death, and 4(12.5%) patients had maternal complications.
- In majority of the patients BP control was achieved within a week
- Most of the women required hospitalisation for 2 to 3 weeks with a minimum of 7 days and a maximum of 31 days.
- Mean of postpartum hospital stay was 16 days.
- Prolonged hospitalisation in most of the women was for baby sake

CONCLUSION

Early onset severe preeclampsia is associated with significant maternal and fetal complications.

Early booking is essential for better maternal and fetal outcome.

Decision regarding termination of pregnancy has to be taken based on both maternal and fetal factors. In case of severe uncontrolled blood pressure with complications, termination should be done irrespective of fetal maturity. Good NICU improves the fetal prognosis.

In selected cases expectant management in a tertiary care centre limit the impact of serious maternal and fetal complications.

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PROFORMA

NAME: **AGE:** **IP NO:**
ADDRESS: **DOA:**
DOD:

BOOKED/UNBOOKED: **LMP:**
EDD:

PRESENTING COMPLAINTS

H/O Amenorrhea

H/O Headache/epigastric pain/ blurring of vision/decreased Urine output.
H/O Bleeding Pv/ Draining PV
H/O Perception Of Fetal Movements.

MENSTRUAL HISTORY

Age at menarche;
Menstrual cycles:
LMP: EDD:

MARITAL HISTORY

Married Since:
Consanguineous/ non consanguineous

OBSTETRIC HISTORY

GRAVIDA: PARA: ABORTION:
LIVE: DEATH:
Immunisation:
Previous Pregnancy:
Present Pregnancy:
1ST Trimester:
2ND Trimester:
3RD Trimester:
Last child birth:

PAST HISTORY

Renal Disease Liver Disease/ Diabetes/ Epilepsy/RHD/Thyroid

Disease/Bronchial Asthma

FAMILY HISTORY

Hypertension:
Diabetes:
Patient's mother had preeclampsia / eclampsia:

PERSONAL HISTORY

DIET: SLEEP: BLADDER: BOWEL:

GENERAL EXAMINATION

HT: WT: BMI:
TEMP: BP: PR: RR:
Pallor / Icterus / Pedal Edema
CVS: Breast:
RS: Thyroid:

PER ABDOMEN:

Size of uterus(in weeks):
Acting/not acting:
Presenting part:
Engaged/ unengaged:
FHR:

PER VAGINAL EXAMINATION:

INVESTIGATIONS

URINE INVESTIGATIONS:
Urine Albumin: Sugar: Deposits:
BLOOD INVESTIGATIONS:
HB: PCV: Platelets:
TC: DC:
Blood Sugar:
Blood Urea:
Serum Creatinine:
Serum Bilirubin:
SGOT:
SGPT:
Serum Uric Acid:
Serum T.Proteins:
Serum Albumin:
Serum Electrolytes:

FUNDUS:
ULTRASOUND OBSTETRICS:
FETAL DOPPLER:

TREATMENT GIVEN:

ANTI HYPERTENSIVES;
STEROIDS:
MgSO4:
OTHER:

MODE OF DELIVERY:

Spontaneous / Induced:
If Induction
Mode of induction:
Time of induction:
Induction - delivery interval:
Duration of Labour
1ST Stage: Liquor:
2ND Stage: Placental Weight:
3RD Stage:
Vaginal delivery:
Caesarean section:
If Caesarean, Indication:
Placenta weight:

COMPLICATIONS DURING DELIVERY

NEONATE

Alive / death:
Term / preterm:
Sex:
Apgar 1 Min: 5 Min:
Weight:

POST PARTUM FOR 72 HRS

Consciouness:
Temp: PR: BP: RR:
Urine Output:
BP Normalises in:
CVS: RS: CNS:
P/A: P/V:

MATERNAL OUTCOME

Alive / Death: Morbidity:
If Dead, Cause Of Death:

FETAL OUTCOME

Alive / Death: Morbidity:
IUFD: IUGR:
Still Birth:

NO OF DAYS OF POST PARTUM HOSPITALISATION:

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
1	Devi	26	36259	P	B	IV	-	32+1	-	+	O	No	Yes	+	25	36+2	LSCS	F	9	1.85	Normal	Normal	5	10
2	Roja	25	865	M	B	IV	+	30	-	-	O +P	Yes	No	-	-	30	LSCS	M	6	900	Normal	DEATH	3	16
3	Samundeshwari	23	1801	P	B	IV	-	26	-	-	O	No	No	-	-	26+1	VD	M	0	550	Normal	IUFD	3	7
4	Mohana	26	2278	M	B	V	-	33	Gr I	-	O +P	Yes	No	-	-	33	LSCS	M	7	1.5	Eclampsia	Normal	4	20
5	Priya	27	2362	M	B	V	+	33+4	-	-	O	Yes	Yes	+	2	33+6	LSCS	M	9	1.9	Normal	Normal	2	10
6	Sathya	19	2473	P	B	V	-	28	-	-	O	No	No	-	-	28	LSCS	M	0	600	Eclampsia	Stillborn	5	10
7	Suguna	36	1713	M	B	IV	+	30	-	-	O	No	Yes	+	14	32	LSCS	M	8	1.25	Normal	IUGR/RDS	4	25
8	Sangeetha	26	2995	P	B	V	-	33+3	Gr I	-	O +P	Yes	Yes	-	-	33+3	LSCS	M	9	2	Normal	Normal	3	9

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
9	Revathy	23	4289	P	B	V	-	32	Gr I	-	O +P	Yes	No	-	-	32	LSCS	M	6	1.2	Eclampsia	DEATH	14	20
10	Pattu	18	3944	P	B	V	-	27	-	-	O	No	Yes	+	5	27+5	LSCS	M	6	1.4	Eclampsia	IUGR/RDS	7	22
11	Deepa	23	4537	P	B	IV	-	32	Gr I	-	O	Yes	Yes	-	-	32	LSCS	M	8	1.9	Wound infection	Normal	5	12
12	Tamilselvi	29	5731	M	B	V	-	33	-	-	O +P	Yes	Yes	-	-	33	LSCS	M	8	1.6	Normal	IUGR	6	14
13	Chithra	32	6176	M	B	IV	+	33	-	-	O	Yes	Yes	+	1	33+1	LSCS	M	8	1.75	Normal	Normal	5	23
14	Kalaivani	26	6247	M	B	V	-	32	-	-	O	Yes	Yes	-	-	32	LSCS	M	7	1.3	Normal	RDS/HIE2	4	15
15	Premavathy	31	6408	P	B	IV	-	32	-	-	O	No	No	-	-	32+1	LSCS	M	7	1.6	Normal	Normal	12	18
16	Bharani	29	6405	P	B	V	-	27	Gr II	-	O	Yes	No	-	-	27+1	LSCS	M	0	500	ARF	IUFD	20	28

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
17	Vimala	24	7673	P	B	IV	-	33	-	-	O	Yes	No	-	-	33	LSCS	M	8	2.2	Normal	Normal	4	11
18	Gayathri	23	7241	P	B	IV	-	27	-	+	O+P	No	Yes	+	8	28+1	LSCS	F	7	1.2	Normal	RDS	6	28
19	Manjula	35	8119	M	B	IV	-	28	-	-	O	No	Yes	+	1	28+1	LSCS	M	8	1	Normal	Normal	12	35
20	Sivaranjani	21	8180	P	B	V	+	33+6	Gr I	-	O	Yes	No	-	-	33+6	LSCS	M	8	2	Normal	Normal	6	10
21	Selvi	19	10002	P	UB	IV	-	28	Gr I	-	O+P	Yes	No	-	-	28	LSCS	M	4	900	Wound infection	DEATH	5	18
22	Rajeshwari	19	10288	P	B	V	-	33+5	-	-	O+P	Yes	No	-	-	33+5	LSCS	M	8	2.3	Normal	Normal	3	10
23	Ramya	27	4756	M	B	V	-	32	-	-	O	No	No	-	-	32	LSCS	M	7	1.3	Abruption	RDS/HIE 1	4	25
24	Tamilarasi	37	4835	M	B	IV	+	31	-	+	O	No	Yes	+	21	34	LSCS	F	7	1.2	Normal	Normal	5	19

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
25	Vijayalakshmi	24	5008	P	B	IV	-	33	Gr II	-	O	No	No	+	5	33+6	LSCS	M	7	1.45	Normal	Normal	6	22
26	Saroja	25	5411	P	B	V	-	30+2	-	-	O	Yes	No	-	-	30+2	LSCS	M	0	950	Normal	IUFD	5	12
27	Selvarani	26	11036	M	B	IV	+	32	-	+	O	No	Yes	+	7	33	LSCS	F	8	1.25	Normal	IUGR	6	18
28	Sridevi	18	12000	P	B	IV	-	33+1	-	-	O	No	Yes	+	5	33+6	LSCS	M	8	2	Normal	Normal	4	8
29	Vinodhini	18	12951	P	B	V	-	30	Gr I	-	O	No	No	-	-	30+1	VD	F	0	750	Normal	IUFD	3	7
30	Saranya	20	13849	P	B	IV	-	32	-	-	O	No	Yes	+	2	32+2	LSCS	M	9	2	Normal	Normal	6	12
31	Amudha	21	13966	P	B	IV	-	32	Gr II	-	O+P	Yes	No	-	-	32	LSCS	M	9	2.2	Wound infection	Normal	4	15
32	Sathya	23	14332	M	B	IV	+	32	-	-	O+P	Yes	No	-	-	32	LSCS	M	6	1.3	Normal	IUGR	6	17

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
33	Zeenua Vahan	19	14784	P	B	V	-	31	-	-	O	No	Yes	+	4	31+4	LSCS	M	8	1.8	Normal	Normal	5	10
34	Punitha	20	15208	P	B	IV	-	33	-	-	O	No	No	-	-	33	LSCS	M	8	1.8	Abruption	RDS	4	12
35	Sumathy	21	32057	P	B	IV	-	33	-	-	O	Yes	No	-	-	33+1	LSCS	M	8	1.8	HELLP / DIC	Normal	7	28
36	Kanniyamal	28	15765	P	B	V	-	32+4	-	-	O	No	Yes	-	-	32+5	VD	M	4	1.8	PP Eclampsia	RDS	20	35
37	Sumithra	28	15623	M	B	IV	-	30	Gr I	-	O	Yes	No	-	-	30	LSCS	M	4	880	Normal	DEATH	4	12
38	Sumathy	20	15329	P	B	V	-	28+4	Gr II	-	O+P	Yes	No	-	-	28+4	VD	F	0	800	Normal	IUFD	6	10
39	Andal	32	15735	M	B	IV	-	30	-	-	O	No	Yes	+	7	31	LSCS	M	8	1.65	HELLP	Normal	5	20
40	Meena	29	16632	P	B	V	+	33	Gr II	-	O+P	Yes	No	-	-	33	LSCS	M	7	2	Abruption	Normal	3	12

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
41	Radha	23	16660	P	B	IV	-	30+1	-	-	O	Yes	Yes	-	-	30+2	VD	M	5	1.2	Normal	RDS / IUGR	5	20
42	Kalaiarasi	22	16598	P	B	V	-	33	-	-	O	No	Yes	+	5	33+5	LSCS	F	8	2	Normal	Normal	4	12
43	Godavani	28	17415	M	B	IV	-	30	Gr I	-	O+P	Yes	No	-	-	30	VD	F	IUFD	800	Normal	IUFD	3	10
44	Anjalai	18	17708	P	B	V	-	33	-	-	O	Yes	Yes	-	-	33	LSCS	M	5	2	Normal	HIE2 / Asphyxia	14	18
45	Sudha	23	17715	P	B	IV	-	32+1	Gr II	-	O+P	Yes	No	-	-	32	VD	M	IUFD	1.6	Normal	IUFD	4	7
46	Usha	25	16832	P	B	IV	-	33+5	-	-	O	Yes	No	-	-	33+5	LSCS	M	9	2.3	Wound infection	Normal	6	22
47	Devi	25	18606	P	B	V	-	33	-	-	O	Yes	No	-	-	33	VD	M	3	1.6	Normal	Death	5	10
48	Radha	28	19227	M	B	IV	+	32	Gr I	-	O+P	Yes	No	-	-	32	VD	M	9	2	Normal	Normal	11	14

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
49	Sudha Sree	20	19361	P	B	IV	-	32	-	-	O	Yes	No	-	-	32+1	VD	M	6	1.2	Normal	IUGR	6	22
50	Thiruveni	23	20403	P	B	V	+	33+4	Gr I	-	O	No	No	-	-	33+4	VD	F	0	1.2	Normal	IUFD	4	9
51	Selvi	36	20638	M	B	IV	+	28	-	-	O	No	Yes	+	14	30	LSCS	M	7	1.6	Normal	IUGR	12	20
52	Nagammal	23	21301	P	B	V	-	33+5	Gr I	-	O	Yes	No	-	-	33+5	LSCS	M	0	1.6	Abruption	IUFD	7	14
53	Rani	23	21155	P	B	V	-	31	-	-	O+P	Yes	No	-	-	31	LSCS	M	5	1.4	Eclampsia	RDS / HIE2	4	15
54	Renuka	23	21311	P	B	IV	-	33	-	-	O+P	Yes	No	-	-	33	VD	M	7	1.6	Eclampsia	Normal	6	17
55	Arivuselvi	21	21573	P	B	IV	-	31	-	-	O	Yes	Yes	-	-	31+1	Lscs	M	8	1.5	Normal	Normal	31	12
56	Valli	21	22014	P	B	V	-	30	Gr I	-	O	No	No	-	-	30	VD	F	IUFD	1.05	Normal	IUFD	4	10

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
57	Gowri	32	22109	M	B	IV	+	33	-	-	O	No	Yes	+	3	33+3	LSCS	M	8	2.1	ARF	Normal	7	25
58	Rajathi	23	23095	P	B	IV	+	33	Gr II	-	O+P	Yes	No	-	-	33	LSCS	M	8	1.6	Eclampsia	IUGR	5	14
59	Kanikkai	25	23526	M	B	V	-	30	-	+	O	No	Yes	+	5	30+5	LSCS	F	7	1.2	Normal	IUGR	6	25
60	Shanthy	40	22877	P	B	IV	-	33	-	-	O	Yes	No	-	-	33	LSCS	M	8	1.5	Normal	RDS / HIEI	5	12
61	Esther	26	24144	P	B	IV	-	32	-	-	O	No	No	-	-	32	VD	F	0	1	Normal	IUFD	6	11
62	Amudha	29	25411	M	B	V	+	33	-	-	O	Yes	Yes	-	-	33	LSCS	M	7	1.7	Normal	Normal	5	12
63	Jayalakshmi	28	25621	M	B	V	+	33	-	-	O	Yes	No	-	-	33	LSCS	M	8	1.6	Atonic PPH	IUGR	6	14
64	Latha	29	25822	M	B	IV	-	33	-	-	O	No	No	-	-	33	LSCS	M	0	1.4	Abruption	IUFD	3	13

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
65	Geetha	22	26033	P	B	IV	-	32	-	-	O	Yes	No	-	-	32	LSCS	M	7	1.2	Normal	IUGR	5	14
66	Amudha	29	25411	M	B	IV	+	33	-	-	O	No	Yes	-	-	33	LSCS	M	7	1.7	Normal	Normal	7	15
67	Jayalakshmi	28	25621	M	B	V	-	33	-	-	O	Yes	No	-	-	33	LSCS	M	8	1.6	Wound infection	IUGR, RDS	6	21
68	Latha	32	25822	M	B	IV	+	31	Gr I	-	O	No	No	-	-	31	LSCS	M	0	1.2	Abruption	IUFD	5	13
69	Geetha	22	26033	P	B	IV	-	32	-	-	O	No	Yes	+	7	33	LSCS	M	8	1.6	Normal	Normal	7	15
70	Ansarbee	23	26589	P	B	IV	-	32+1	Gr I	-	O+P	Yes	No	-	-	32+1	VD	M	6	1.4	Normal	IUGR / RDS / HIEI	3	14
71	Divya	21	26832	M	B	IV	+	26+4	-	-	O	No	Yes	+	20	29+3	VD	M	5	1	Normal	DEATH	8	22
72	Kasthari	27	26629	M	B	IV	-	32	Gr I	-	O	No	No	-	-	32	VD	M	0	1.3	Abruption	IUFD	4	26

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
73	Rajeshwari	31	26328	M	B	V	+	30	-	-	O+P	Yes	Yes	-	-	30+1	VD	F	7	1.03	Normal	Normal	7	18
74	Maheswari	27	24439	P	B	IV	-	32+5	-	-	O	No	No	-	-	32+5	VD	F	0	1.5	Normal	IUFD	3	10
75	Mary	19	27157	P	B	IV	-	33	-	+	O	No	Yes	+	3	33+3	LSCS	F	7	1.75	Normal	Normal	4	15
76	Prasanna	21	27321	M	B	IV	+	33	Gr I	-	O+P	Yes	No	-	-	33	LSCS	M	0	1.8	Abruption	IUFD	3	20
77	Chithra	32	28321	P	B	IV	+	25	-	-	O	Yes	No	-	-	25	VD	M	0	800	Normal	IUFD	4	13
78	Vanitha	25	28360	P	B	IV	-	33+5	-	-	O+P	Yes	No	-	-	33+5	LSCS	M	8	1.5	Normal	RDS / Seizures	5	20
79	Jayashree	25	28168	P	B	IV	-	32	Gr I	-	O	Yes	No	-	-	32+2	LSCS	M	7	1.2	DIVC / Relaprotomy	Death	12	35
80	Durga	19	28458	P	B	IV	-	33	-	-	O	Yes	No	-	-	33	LSCS	M	8	1.95	Wound infection	Normal	7	22

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
81	Anitha	22	28329	P	B	IV	-	32	-	-	O	Yes	No	-	-	32	VD	F	IUFD	1	Normal	IUFD	5	10
82	Vanaja	24	28720	M	B	IV	-	33	-	-	O	Yes	No	-	-	33	LSCS	M	8	1.5	Normal	RDS / HIEI	6	22
83	Lakshmi	22	29578	M	B	V	-	33+4	Gr II	-	O+P	Yes	No	-	-	33	LSCS	M	4	1.4	HELLP, DIVC	Death	8	20
84	Srividhya	25	30193	M	B	IV	+	31+5	-	-	O	Yes	No	-	-	31+5	VD	M	6	1.2	Normal	IUGR / Septicemia	6	25
85	Indhumathi	21	30232	M	B	IV	-	33+4	-	-	O	Yes	No	-	-	33+4	LSCS	M	8	1.5	Normal	Normal	4	15
86	Kanagavalli	21	30805	M	B	IV	+	33+5	-	-	O	Yes	No	-	-	33+5	VD	M	7	1.7	Eclampsia	Normal	5	13
87	Saranya	27	28589	P	B	IV	-	33	Gr II	-	O	Yes	No	-	-	33	LSCS	M	8	2	Death	Normal		Admission - Death Inteval 24 hrs
88	Mala	27	32385	M	B	V	-	33+5	-	-	O+P	Yes	No	-	-	33+5	LSCS	M	3	1.6	Death	Death		36

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
89	Sharmila	22	18775	P	B	IV	-	30	Gr I	-	O	Yes	No	-	-	30	LSCS	M	0	1	Death	Death		12
90	Dhanalakshmi	19	7587	P	B	V	-	31	-	-	O	Yes	No	-	-	31	LSCS	M	0	1.2	Death	Stillborn		5
91	Jothi	34	3586	M	B	IV	+	32	Gr II	-	O+P	Yes	No	-	-	32	LSCS	M	0	1.25	Death	IUFD		1d 6hrs
92	Sethamilselvi	28	385	P	B	V	-	30	-	-	O	Yes	No	-	-	30	VD	M	0	1	Death	IUFD		1d 12hrs
93	Karthika	23	19934	P	B	III	-	28+4	-	-	O	Yes	No	-	-	28+4	VD	M	3	900	Normal	Death	7	11
94	Monika	20	32093	P	B	IV	-	31+3	-	-	O+P	Yes	No	-	-	31+3	LSCS	M	0	1.3	Normal	Stillborn	4	14
95	Dhanalakshmi	24	32079	P	B	V	-	33+5	-	-	O	Yes	Yes	-	-	33+6	LSCS	M	8	1.7	Normal	Normal	5	14
96	Kanagavalli	21	30895	M	B	IV	+	33+3	Gr I	-	O+P	Yes	Yes	-	-	33+4	VD	M	6	1	Normal	DEATH	4	7

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
97	Usha	25	16832	P	B	V	-	33+5	-	-	O	Yes	No	-	-	33+5	LSCS	M	7	1.5	Eclampsia	IUGR / RDS	5	14
98	Ramya	21	32760	P	B	V	-	29+1	-	-	O+P	Yes	Yes	-	-	29+2	LSCS	M	8	1.25	Normal	Normal	4	16
99	Gomathi	32	35695	M	B	IV	+	32+2	Gr I	-	O	No	Yes	-	-	32+2	VD	M	0	700	Normal	IUFD	3	7
100	Saranya	19	12368	P	B	V	-	28	-	-	O	No	Yes	+	4	28+4	LSCS	M	6	1.05	Normal	RDS, Septicemia	5	21
101	Saraswathi	22	16832	P	B	IV	-	30+2	-	-	O	Yes	Yes	+	3	30+5	LSCS	M	7	1.3	PP Eclampsia	RDS	6	14
102	Vimala	18	17829	P	B	V	+	27+2	-	-	O	Yes	No	-	-	27+2	VD	M	4	1	Eclampsia	Death	5	14
103	Vaideki	35	18321	M	B	IV	+	33	-	-	O	Yes	Yes	-	-	33	VD	M	7	1.6	Normal	RDS, CPAP	4	20
104	Malathi	23	16324	P	B	V	-	32	-	-	O	Yes	No	-	-	32	LSCS	M	6	1.5	Eclampsia	Normal	6	20

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105	Niranjana	25	18392	P	B	IV	-	33+3	-	-	O	Yes	No	-	-	33+3	LSCS	M	5	1.4	Normal	IUGR	10	16
106	Banu	19	19342	P	B	V	+	32	-	-	O	Yes	Yes	-	-	32+1	LSCS	M	6	1.5	Normal	HIE2 / Septicemia	5	18
107	Chandra	24	20012	P	B	IV	-	33+5	-	-	O	Yes	No	-	-	33+5	LSCS	M	5	1.4	Normal	RDS	6	21
108	Shanthi	20	20132	P	B	IV	-	28	-	-	O+P	No	Yes	+	10	29+3	VD	M	6	1	Normal	RDS, HIE2	5	25
109	Shyalaja	27	8679	P	B	V	-	32	-	-	O	Yes	No	-	-	32+1	VD	M	7	1.6	Normal	Normal	5	20
110	Dhanam	20	6623	P	B	IV	-	33+1	-	+	O	No	Yes	+	3	33+4	LSCS	F	7	1.8	Normal	Normal	4	14
111	Ezhilarasi	34	4329	M	B	IV	+	27	Gr I	-	O	No	No	-	-	27	VD	M	0	900	Abruption	IUFD	4	12
112	Fathima	28	32489	P	B	V	-	32+5	-	-	O	Yes	Yes	-	-	33+6	LSCS	M	7	1.6	Normal	Normal	10	16

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
113	Gomathy	20	23229	P	B	V	-	32+5	-	+	O	No	Yes	+	2	33	LSCS	F	7	1.7	Normal	Normal	7	18
114	Girija	29	23329	P	B	IV	-	33	-	-	O	Yes	Yes	-	-	33+1	VD	M	6	1.5	Normal	Normal	6	21
115	Astalakshmi	23	23369	P	B	V	-	33+5	-	-	O	Yes	Yes	+	2	34	LSCS	M	7	1.6	Normal	Normal	5	16
116	Amudha	24	24321	P	B	IV	-	33	-	-	O	Yes	Yes	-	-	33+1	VD	M	7	1.7	Normal	Normal	3	15
117	Banumathy	32	35829	M	B	V	+	28	Gr II	-	O+P	Yes	No	-	-	28	LSCS	M	5	1	Eclampsia	RDS, HIE	5	14
118	Jaya	27	25889	M	B	IV	-	33+5	-	-	O	Yes	Yes	-	-	33+6	VD	M	8	1.8	Normal	Normal	4	13
119	Kalpana	20	26821	P	B	V	+	26+3	-	-	O	Yes	No	-	-	26+4	VD	M	6	800	ARF	DEATH	10	35
120	Jamuna	33	26991	P	B	V	-	32+2	-	-	O	Yes	Yes	-	-	32+3	LSCS	M	7	1.7	Normal	Normal	4	11

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
121	Kanimozhi	20	27112	P	B	IV	-	33+6	-	+	O	No	Yes	+	5	34+4	LSCS	F	7	1.6	Normal	Normal	5	10
122	Kalai	32	27331	M	B	V	+	33+5	-	+	O	No	Yes	+	2	34	LSCS	F	8	0	Normal	Normal	4	13
123	Madhavi	32	28829	M	B	V	+	31	-	-	O	Yes	No	-	-	31	LSCS	M	6	1.5	Eclampsia	RDS, HIE2	6	14
124	Maheswari	28	28902	P	B	IV	-	30	-	-	O	Yes	No	-	-	30	LSCS	M	0	1.4	Abruption	IUFD	5	13
125	Latha	33	29201	P	B	V	-	32	-	-	O	Yes	No	-	-	32	VD	M	6	1.4	Wound infection	IUGR	5	14
126	Revathy	26	29323	P	B	V	-	31+5	-	-	O	Yes	No	-	-	31+5	LSCS	M	3	1.7	Abruption	Death	4	12
127	Priya	20	29414	P	B	IV	+	28	-	-	O+P	Yes	Yes	-	-	28+2	LSCS	M	5	1.1	Normal	HIE2, Septicemia	6	14
128	Subha	25	29432	M	B	V	-	32	-	-	O	Yes	Yes	-	-	32+1	VD	M	6	1.5	Normal	Normal	4	15

S.No	Name	Age in Years	IP No	Parity	Booked / Unbooked	Socio Economic Status	Antenatal Risk factors +/-	Gest age at Diagnosis	Fundas	Doppler changes	Anti hypertensives oral (O)/ Parenteral (P)	Mgso4	Steroids	Expectant management +/-	No. of days gained	Gest Age at delivery	Mode of delivery	Indication for termination Maternal / Fetal	APGAR	Birth Weight	Maternal Outcome	Fetal outcome	Postpartum BP Control	Post partum hospitalisation
129	Lalitha	20	29621	P	B	IV	-	31+6	-	-	O	Yes	Yes	-	-	32+1	LSCS	M	7	1.5	Normal	Normal	6	21
130	Pappu	21	29843	P	B	IV	-	32+6	-	-	O	Yes	No	-	-	32+6	LSCS	M	7	2	Normal	Normal	4	15
131	Shoba	36	29991	M	B	V	+	26+3	-	-	O+P	Yes	No	-	-	26+3	VD	M	6	1	PP Eclampsia	HIE2, Septicemia	12	22
132	Leelavathi	19	30102	P	B	V	+	33+1	-	+	O	Yes	No	-	-	33+1	LSCS	M	7	1.8	Normal	Normal	5	14
133	Sandhya	23	30221	P	B	IV	-	33+3	-	-	O	No	Yes	+	4	34	LSCS	F	7	1.7	Normal	Normal	6	13
134	Santhi	34	30289	M	B	V	-	33+6	-	-	O	Yes	No	-	-	33+6	LSCS	M	6	1.8	Normal	Normal	3	16
135	Pavithra	28	31832	P	B	IV	+	27+5	-	-	O	No	No	-	-	27+5	VD	M	0	900	Abruption	IUFD	3	14
136	Lakshmi	20	31419	P	B	IV	-	32+6	-	+	O	No	Yes	+	2	33+1	LSCS	F	7	1.7	Normal	Normal	4	18

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137	Meena	29	31278	M	B	V	+	33+1	-	-	O	Yes	No	-	-	33+1	LSCS	M	7	1.6	Normal	Normal	5	12
138	Renuka	31	32287	M	B	IV	-	33+2	-	-	O	Yes	Yes	-	-	33+3	VD	M	7	1.5	Normal	Normal	6	10
139	Uma	18	32389	P	B	V	-	33+5	-	-	O	Yes	Yes	-	-	33+6	VD	M	7	1.6	Normal	Normal	8	17
140	Neelavathi	20	32410	P	B	V	-	33+6	-	-	O	Yes	No	-	-	33+6	VD	M	7	1.5	Normal	Normal	10	12
141	Fathima	26	32520	P	B	IV	+	27+6	-	-	O+P	Yes	No	-	-	27+6	LSCS	M	0	900	Eclampsia	Stillborn	6	13
142	Usha	31	32589	M	B	V	+	32	-	-	O	Yes	No	-	-	32	VD	M	6	1.3	Atonic PPH	RDS / HIEI	4	15
143	Pankajam	27	33321	P	B	IV	-	33+5	-	-	O	Yes	Yes	-	-	33+6	VD	M	7	1.3	Atonic PPH	IUGR	3	14
144	Chandra	31	34381	P	B	V	-	33+3	-	-	O	Yes	No	-	-	33+3	LSCS	M	6	1.4	Wound infection	IUGR	4	16

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145	Menaka	20	35621	P	B	IV	-	32+5	-	-	O	Yes	No	-	-	32+5	LSCS	M	3	1.3	Eclampsia	IUGR, HIE2	5	21
146	Kavitha	25	36829	P	B	V	-	33+2	-	-	O	Yes	No	-	-	33+2	LSCS	M	4	1.6	Ahruption	HIE2, Septicemia	4	16
147	Jothi	31	36921	M	B	IV	+	28	-	-	O	No	Yes	+	7	29	VD	M	6	1.1	Normal	DEATH	6	18
148	Nisha	17	37820	P	B	V	-	33+4	-	-	O	Yes	No	-	-	33+4	LSCS	M	0	1.2	Ahruption	IUFD	3	13
149	Hema	23	38921	P	B	IV	-	32+6	-	-	O	Yes	No	-	-	32+6	LSCS	M	5	1.5	Eclampsia	HIE2 / IUGR	7	13
150	Megala	20	39981	P	B	V	-	33	-	-	O	Yes	No	-	-	33	LSCS	M	7	1.6	Eclampsia	RDS, Septicemia	8	20

KEY TO MASTER CHART

P	Primigravida
M	Multigravida
B	Booked
UB	Unbooked
O	Oral antihypertensive
P	Parenteral antihypertensive
VD	Vaginal delivery
LSCS	Lower segment caesarean section
M	Maternal
F	Fetal
IUGR	Intra Uterine Growth Restriction
IUFD	Intra Uterine Fetal Demise
RDS	Respiratory distress syndrome
HIE	Hypoxic ischaemic enchelopathy

ABBREVIATIONS

CRP	C Reactive protein
BMI	Body Mass Index
MAP	Mean Arterial Pressure
HCG	Human Chorionic Gonadotrophin
MSAFP	Maternal Serum alpha fetoprotein
HELLP	Hemolysis, Elevated Liver Enzymes, Low Platelet count
IUGR	Intra Uterine Growth Restriction
GA	Gestational Age
ARF	Acute Renal failure
DIVC	Disseminated Intravascular Coagulation
IUFD	Intra Uterine Fetal Demise
PNM	Perinatal Mortality
SB	Stillbirth
PND	Perinatal death
RDS	Respiratory distress syndrome
HIE	Hypoxic ischaemic enchelopathy
PP ECLAMPSIA	Post partum eclampsia